JAN 30 1932

VOL. XXV., NO. 3 JANUARY, 1932.

PROCEEDINGS of the ROYAL SOCIETY OF MEDICINE

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Section of Urology.

President—Mr. RALPH THOMPSON, Ch.M.

[*October* 22, 1931.]

Electro-surgery of the Bladder Neck.

By CLYDE W. COLLINGS, M.D., F.A.C.S.
(*New York*).

CERTAIN types of bladder-neck obstruction may be efficiently excised through the cysto-urethroscope, by the cutting high-frequency current.

Indications for operation.—(1) Fibrous contracture or bar of the bladder neck; (2) Obstructing scar following prostatectomy; (3) Carcinomatous bar (from prostatic carcinoma); (4) Small median or small intra-urethral lobes.

Contra-indication.—Benign enlargement of the lateral lobes of the prostate. Patients with this type of obstruction are better relieved by prostatectomy.

I have tried trans-urethral surgery upon enlarged lateral lobes, but thus far have failed to get satisfactory results. In my hands large lateral lobes bleed very readily, and I find it difficult to whittle out enough tissue to do any good. Some surgeons in America are enthusiastically biting out pieces of enlarged lateral lobes and claim very good results. Caulk has used his punch on the same patient as many as eight or nine times.

The advantages of the operation are: (1) The efficient excision of certain obstructions at the bladder neck by a trans-urethral method; (2) it is under direct vision at all times; (3) primary bleeding is unimportant—late secondary bleeding is nil; (4) The patient is relieved of a grave lesion, by a minor procedure, without endangering life.

The author's cysto-urethroscope is used because of the continuous irrigation which it provides. The field of vision is very large, the electrode and operative area being in view at all times.

The electrical apparatus employed is a spark-gap machine similar in principle to the battery used to fulgurate bladder tumours. The high-frequency oscillations are "stepped up" some fourteen or fifteen times faster—giving a current that cuts instead of coagulates. By microscopic examination we have determined that heat penetrates and destroys tissue 1 or 2 mm. beyond the cut.

History of trans-urethral operations.—Since the days of Bottini urologists have concerned themselves with a trans-urethral operation for relieving small fibrous obstruction. This cautery caused considerable bleeding as the thick slough separated a week or two later. Incontinence was another complication.

Keyes states, "Young has given us a urethral punch wherewith to bite out pieces of the bladder neck, and continues to perform this operation undismayed by the profuse hæmorrhage that often results." Like Young, Caulk uses his punch without the aid of the eye.

Soon after Beer brought out the high-frequency apparatus, he fulgurated a median bar; twenty-two days later the patient had an almost fatal hæmorrhage, controlled only by suprapubic operation.

Since April, 1923, I have been using the cutting current through the cysto-urethroscope at New York University and Bellevue Hospital. I have operated upon some 700 patients in my clinic and private practice since that time. There have been no operative deaths.

I wish at this time to summarize my results upon eighty private patients operated upon since 1926.

Age incidence.—The youngest patient was aged 28 (a congenital bar), and the oldest 92; the average age of all patients was 54.

Symptoms.—Frequency was the chief primary complaint of fifty patients; burning in twenty-seven, and inability to void in nine. Of secondary consideration eleven mentioned difficulty; nine, pain across the back; nine, pain in the perineum; eleven, discharge from the urethra; seven, hæmaturia; six, small stream; five, dribbling; five, suprapubic pain; three, urgency; two, stones; two, chills and fever; and at least one patient complained of inability to empty bladder, mental fog, spasms, weakness, pain in rectum and partial impotence.

The following operations had been performed on this group previous to my operation: stricture, two patients; litholapaxy, three patients; prostatectomy, three patients; fulguration of median bar, one patient; orchidectomy, one patient, and Young punch, one patient.

The average duration of symptoms before operation was four years and three months; the shortest period two weeks, and the longest twenty-two years. The residuum before operation varied from zero to 495 c.c., the average being 90 c.c.

The prostate was described as being normal in size and firm throughout in twenty-five patients; slightly enlarged in thirty-eight; moderately enlarged in ten; carcinomatous in four; and removed previously by prostatectomy in three.

The functional phenolsulphonephthalein test was usually quite good, 28 to 75 per cent., average 40 per cent., in two hours after intramuscular injection. The non-protein nitrogen and creatinin of the blood rarely showed more than a slight retention.

The cystoscopic diagnosis.—As the cysto-urethroscope is passed through the bladder neck, depression of the ocular end is required to get over the fibrous bar. Often a distinct jump is felt as the instrument slips by the obstruction. In patients with symptoms over a period of years, trabeculation of the bladder is often noted. There is a tilting of the trigone and a definite *bas fond*. As the urethroscope is withdrawn into the posterior urethra the floor of the bladder neck seems to rise as a curtain. Pushing the instrument back towards the bladder, a "stone wall" is encountered, the obstruction can only be passed if the ocular end is again depressed. In the collar contracture cases the bladder neck, as viewed from the prostatic urethra, looks as if it had been drawn together with a purse string.

Operation.—Caudal anæsthesia (50 c.c. of 1 per cent. novocain) in the sacrum is usually sufficient; spinal or gas is used at times.

The cysto-urethroscope is passed into the bladder; the inflow and outflow faucets are carefully regulated. This gives the following advantages: (1) One may work without the interruption of changing the distending medium of the bladder. (2) A certain fixed distension of the posterior urethra and bladder may be maintained.

The electrode knife is engaged on the bladder neck at the position of 6 on the clock dial. The current is turned on and violent bubbling is noted. The protein molecules may be said to be exploded by the jostling of the high-frequency oscillations. The urethroscope and electrode are slowly pulled back *en masse* until the verumontanum appears. A white furrow, about 2 mm. deep, is seen. The instrument, with the electrode in the furrow, is then pushed forward and through the bladder neck. Working back and forth in this manner the groove is gradually widened and deepened. Cut until you are certain the last obstructing fibrous band has been sawed into. One can, from the verumontanum, look down a deep valley (perhaps 1·5 cm. deep) and see the base of the bladder. Persist in your efforts until you are satisfied that the patient has a wide-open bladder neck. If this is accomplished the patient is cured. We were too timid when performing our first operations, and did not cut deeply enough. But a second operation cured these few patients. One never cuts into the rectum (it is about 1·5 cm. away when the operation is completed).

By turning the knife sideways, one engages the blade on the bladder neck at 5 o'clock. Cut downward and backward towards the verumontanum until the

intervening tissue is whittled away. The procedure is repeated on the opposite side at 7 o'clock. These two lateral incisions merely widen the furrow. The incision which really does the trick of relieving the patient is the deep 6 o'clock cut.

The operation can be slowly and precisely performed in about twenty minutes.

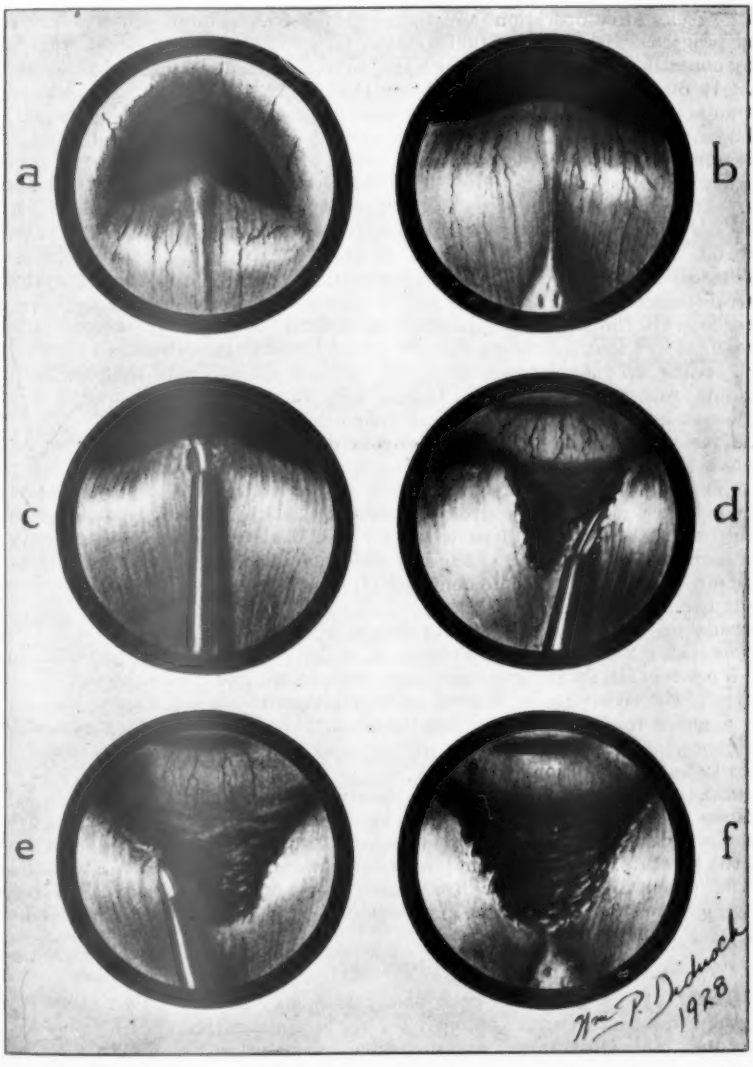

FIG. 1.—(a) Prostatic bar on floor of bladder neck. (b) "Close up" of prostatic bar. (c) Electrode cutting at 6 o'clock on the clock dial. (d) Cutting at 5 o'clock. (e) Cutting at 7 o'clock. (f) The completed operation

After-care.—The indwelling catheter is usually tied in the urethra for twelve hours or so after the operation. If the urine is pink it is left in until the urine becomes clear. Patients with a large residuum or decreased function have catheter drainage for a longer post-operative period. As a rule patients are able to void after removing the catheter. Occasionally a patient may require catheterization for a day or two. As passing a catheter incites bleeding, try not to pass one.

Two weeks after operation a catheter is passed, residuum observed, and the bladder irrigated with a 1 to 5,000 solution of acriflavine. Any slight infection is thereby controlled. This is repeated about once a week and a decrease in residuum noted. It often takes seven or eight weeks before the maximum effects of the operation are obtained, due possibly to the post-operative œdema of the bladder neck and the partial recovery of the normal tone of the chronically distended bladder.

Hæmorrhage from the incisions at the time of operation was nil in fifty-two of the eighty patients of this group. The blood-vessels are sealed as they are cut across. To be sure there is some bleeding from pushing the instrument back and forth in the urethra. In twenty-eight cases, blood-tinged urine with small clots was passed, off and on, for the first week or so. Rarely is it necessary to use an indwelling catheter for bleeding. I have never had to perform a suprapubic cystotomy to control hæmorrhage.

Results.—Of the fifty patients who complained of frequency before operation, thirty-four stated that they were entirely relieved, and sixteen that they were normal by day, getting up once or twice at night. Eleven patients said that the feeling of obstruction was entirely gone. Patients with burning before operation were all relieved save four, and these stated that their burning was only slight.

The residual urine in sixty-five patients *after* operation was recorded as nil; three had a few c.c.; three 15 c.c.; two 20 c.c.; three 30 c.c.; one 45 c.c.; and three amount unknown (patients who could not be traced). This residuum record was made from a few weeks to five years after operation. My first patient, operated upon eight years ago, continues to empty his bladder without symptoms. By urethroscopy his bladder neck appears as wide open as on the day of operation.

The nine patients with acute complete retention were able to void after removal of the indwelling catheter.

Seventy per cent. of these patients were discharged from the hospital in twenty-four hours after operation; 15 per cent. in seventy-two hours; 6 per cent. in five days; 6 per cent. in seven days, and 2 per cent. before two weeks.

Three of the eighty patients developed epididymitis.

As I never injure the triangular ligament, I have never had a patient with incontinence. On rare occasions a patient may lose a teaspoonful of urine for a week or two after operation.

Deaths.—There were no immediate post-operative deaths. Six patients died from three weeks to three years after the bar operation, from an intercurrent disease. The earliest death occurred in a man aged 80, operated upon for carcinomatous bar of the prostate. He died from pneumonia, two and a half weeks after discharge from the hospital. The other five deaths were due to typhoid fever, cerebral hæmorrhage, acute cardiac failure, metastasis from prostatic carcinoma, and renal insufficiency respectively.

Summary.

(1) Several types of bladder-neck obstruction may be efficiently relieved by the cutting high-frequency current through the cysto-urethroscope: (*a*) Fibrous bar and contracture. (*b*) Obstructing scar following prostatectomy. (*c*) At times carcinomatous bar (from prostatic carcinoma). (*d*) Small median and intra-urethral lobes.

(2) Lateral lobe enlargement had best be treated by prostatectomy.

(3) The operation is performed under direct vision at all times.

(4) The bleeding at the time and following operation has been unimportant.
(5) The patients are relieved of their residual urine and symptoms.
(6) A clear-cut cystoscopic diagnosis is extremely important.
(7) The patient spends only a day or so in the hospital in being relieved of a serious lesion, without endangering life.

Discussion.—Mr. J. ALBAN ANDREWS said he had practically no experience of this method; his feeling was that in most cases of prostatic obstruction with residual urine the condition of the kidneys was a matter of prime significance, and so a suprapubic tube retained in position for ten days was a very important part of the treatment. But he gathered from the description that the amount of residual urine in most of Dr. Collings' cases was relatively small, averaging 90 c.c., or about 3 oz., and so there could not have been much back pressure on the kidneys. Therefore, regarded mainly as a procedure for relieving symptoms, he thought it would prove a very valuable method. His own experience of it was limited to three occasions, when he tried to relieve a posterior obstruction by the ordinary coagulation methods with the high-frequency current. He had tried by this means to cut away a posterior intra-vesical projection of small size, so as to relieve symptoms in a patient who was adjudged to be too ill from heart disease to be able to endure a suprapubic prostatectomy.

Mr. KENNETH M. WALKER said that he had employed this treatment in 300 cases. He had long ago concluded that a method of this type was invaluable in urology. These electrical treatments had been brought into disrepute in certain quarters, solely because there had been a lack of discrimination in the selection of cases. The method was excellent provided the case was a suitable one, and he was glad that Dr. Collings laid emphasis on the importance of the cystoscopic examination and the selection of cases.

He confessed that he had used coagulation methods in the treatment of general enlargement of the prostate, but he had done so only in those cases in which prostatectomy had been absolutely out of the question. He had been able to relieve patients so that they had gone on for two years, then had the process repeated, carried on for another two years, and then had come for a third treatment.

He had tried various methods of performing this operation. He had used the diathermy punch, of various types, on two or three occasions, an operation very similar to Dr. Collings' with a similar small knife, made for Mr. Swift Joly seven years ago, but in the long run he fell back on the electrode. Perhaps it was not such a rapid method as Dr. Collings', but in certain cases of median bar one found some enlargement of the lateral lobes, and it was impossible to excavate the lateral lobe with the knife. By means of an electrode, however, one could shave off the prominent portion. He was not suggesting that he was using the method throughout in such cases, but sometimes a better result was obtained from diathermizing the most prominent part of a slightly enlarged lateral lobe.

He had not had any cases of troublesome hæmorrhage, but on two or three occasions he had been troubled by sepsis. Had Dr. Collings had any trouble in this respect?

Mr. F. MCG. LOUGHNANE said that there were cases of fibrous prostate with small median lobe enlargement, or with slight enlargement of the lateral lobes, and these were difficult to remove by the suprapubic method, while the perineal method was not altogether satisfactory. Diathermy absolutely destroyed small median lobes, and when there was enlargement of only one lateral lobe, that also could be destroyed by diathermy. The amount of destruction brought about varied with the time taken and the size of the electrodes used. He used an electrode of 16 charrière, and needle electrodes which he was able to stick into the prostatic tissue, and in that way he destroyed all the prominences he wished to. Through the posterior urethroscope that could be done easily. If there was much tissue to be destroyed he did not think the process should be attempted at one sitting, as sloughs took some time to come away, and there was œdema. As many as six sittings might be required for the destruction of all the tissue.

There were certain objections to the diathermy treatment of enlarged prostate. One was that it was sometimes followed by stricture. Some of his cases so treated did well at the time, but eight or nine months later they had returned complaining of trouble, and he found fibrous tissue forming. Sometimes if one destroyed too much at one sitting there was a large slough, and if the bladder was not in a healthy condition considerable cystitis might follow the operation.

The chief advantage of the diathermy treatment was that, if the operator was content with doing a little destruction at a time, there was never need for the patient to be off duty longer than two or three days, so that if the operation were done on a Friday he would probably be ready to resume his activities on the Monday.

He had always found that caudal anæsthesia was satisfactory in these cases.

Mr. T. J. MILLIN said that he had had success with the electrotome in the four types of case mentioned, and had essayed the method in some types of lateral lobe enlargement, but without success in the latter. He had therefore returned to the electro-coagulation method.

He had had no serious case of hæmorrhage, but in one instance it had been necessary to open the patient again because of sepsis. Two years ago a man was operated upon because of symptoms from a calculous prostate. He was 47 years old when seen, and ten years previously had been attending a urological hospital because he was unable to hold his water. A skiagram showed he had considerable middle-lobe projection, with many stones. Electro-coagulation was carried out at one sitting, and in the intervening two years the patient had had no trouble; he had not had to rise in the night and no longer had the pain he formerly felt, and sexual function was unimpaired.

With regard to diathermy in the treatment of generalized prostatic enlargement, nearly three years ago a patient was treated by the electro-coagulation method, and he gave the usual history. For three months he had had to micturate every hour at night and frequently during the day; during the last four days he had been incontinent. After three weeks of preliminary drainage, tests were carried out revealing a bad condition. The blood-pressure was 208 systolic, urea concentration 1·3 to 1·5, and he was a bad risk for an open operation. Coagulation of the prostate was carried out, and a catheter tied in for eighteen days; he voided urine naturally on the nineteenth day, and went home on the twenty-second day. Seven weeks after the operation the residual urine was 2 oz., and the sloughs had not completely separated. The man was seen two years and nine months afterwards, and then the residual urine was less than three-quarters of an ounce, and he passed the whole night without having to micturate. That case bore witness to the value of diathermy in cases in which it would be risky to operate by the open method.

Mr. JOHN EVERIDGE said that he had tried most of the methods which had been advocated and he had struggled for several years to devise a diathermy punch, in collaboration with Mr. Roper, with only fair results. The aim was to construct a punch so that the surgeon could *see* what he was punching, while he was punching.

There were two main difficulties in the way: (1) interference with good visibility from hæmorrhage, and (2) fixation of the prostatic projection, the tendency being for it to be pushed aside during the punching process. After one or two cases of rather troublesome hæmorrhage he had gone back to electro-coagulation of the gland by "spearing" with an electrode which he had devised for the purpose, as a preliminary to punching, in order to devascularize the part of the gland to be removed. This method of "spearing" had proved very successful and experience seemed to point to its eliminating the necessity for punching, in the majority of cases which came within the scope of the operation. The lateral lobes could be dealt with as well as the median. He had only treated some eighty cases with these methods during the last seven years, a small number on which to base an opinion. He found that only a small proportion of his cases in both hospital and private practice came within the scope of the operation.

He strongly advocated preliminary suprapubic drainage in cases where there was considerable residual urine, renal deficiency and bladder sepsis. He felt much safer if this were done, for two reasons: (1) There was far less general reaction, high temperature and all those symptoms recognized as "kidney shock," and (2) should there be hæmorrhage it caused less local disturbance and was more easily combated. A very large proportion of the patients he treated were those who were far too ill to warrant the operation of prostatectomy and a number had already been condemned to permanent suprapubic drainage.

Mr. H. P. WINSBURY-WHITE: One of the chief difficulties that I have found concerning this form of treatment has been in deciding which cases should be dealt with in this way. I have learned to approach certain cases very cautiously. First of all, when there is a considerable degree of bladder infection, especially if there is residual urine, I have found it unsafe to proceed with electro-coagulation of the bladder neck without some preliminary bladder drainage. Often this is adequately secured by the indwelling catheter, but there

are times when a suprapubic cystotomy seems to be indicated. Neglect of these precautions can quite easily lead to pyelonephritis. Then there is the case presenting prostatic symptoms which has no residual urine at all. It has been my experience that no benefit results in such circumstances from coagulation of the bladder neck, even though the patient complains of difficulty of micturition. The third type of case which I call to mind is one in which, on passing a metal sound, obstruction is met in the prostatic urethra. I am talking now of an inflammatory condition of the prostate, one which is often associated with residual urine and cystitis. I invariably find improvement immediately takes place following intermittent dilatation with a metal sound. Occasionally it may be advisable at a later date to cauterize the bladder neck. If this is necessary it is generally accomplished without fear of complications because of the improved condition resulting from the preliminary treatment.

With regard to treating simple prostatic hypertrophy by electro-coagulation, I have learned from experience that the most suitable cases are those in which, on rectal examination, the evidence of hypertrophy is slight or lacking, but endoscopic investigation shows some small localized adenomatous projection, either into the bladder or prostatic urethra. But the one type of case above all others which benefits from the electric cauterization of the vesical outlet is the one in which the prostatic change is predominantly fibrous. The diagnosis is readily made by noting (1) the absence of hypertrophy on rectal examination, (2) that on passing a metal sound the instrument gives a slight jump as the point enters the bladder, and (3) that on depressing the handle there is resistance offered to this movement by the gland.

Concerning instruments, I was impressed by the fact that Dr. Collings makes use of an endoscope giving direct vision from the posterior urethra to the bladder neck. When I first commenced this form of treatment I made extensive use of Joly's posterior urethroscope, because I found I had such an admirable view of the internal urinary meatus from the posterior urethra, but I eventually gave up this instrument as I was not able to use through it the type of electrode which I considered necessary. More recently I have resorted to the Kirwin bladder-neck electro-cautery punch. The electrode has a sharp point with which the tissue to be coagulated can be transfixed. After each successive portion is destroyed, the punching part of the instrument removes the dead tissue from the field of operation.

Mr. SYDNEY G. MACDONALD said that he would like to know how Dr. Collings dealt with post-prostatic obstruction cases when he was unable to pass the instrument. Every urologist met with this type of case. The patient left the hospital with a patent urethra, yet months later he returned with infected urine and difficulty in micturition. In such cases his (the speaker's) own view was that the primary condition was a contraction, and micturition forced down a cuff of mucous membrane, forming a valve.

THE PRESIDENT said that on a number of points his own views differed materially from some of those put forward. He knew nothing about a "fibrous prostate," though he was told that such occurred. In Mr. Millin's case of calculi in the prostate, skiagrams were taken, and he would like to know whether calculi were still present. [Mr. MILLIN: Stones were passed per urethram.]

He knew little about a "prostatic bar," or of post-prostatic stricture, though he had seen three cases of the latter. One of those cases was dealt with by a surgeon who removed an adenoma of the prostate, and, he thought, the whole prostate was removed too. He had had one case in which stricture followed prostate removal, but that man had a stricture before the prostate was removed.

Another point he would make in relation to this question of surgery was the following. He had every sympathy with Dr. Collings and his disciples, but he was not sure that the so-called back pressure on the kidneys was so, in fact; he believed it was a mild teasing or irritation of the bladder setting up chronic Bright's disease, which was often associated with a slight degree of hydronephrosis. There were serious cases in which palliative measures should be employed, rather than cutting down on the bladder and suprapubic drainage.

Dr. COLLINGS, in reply to Mr. Walker's question as to sepsis, said he thought that if the surgeon's efforts were confined to the urethroscope, working on the floor of the bladder neck, there would be no trouble with sepsis. Certain surgeons on the Continent and in America had had cases of sepsis when they had tackled large lateral lobes, and did not carry out much, or any, drainage.

Replying to Mr. Loughnane, he had never seen a case of stricture of the prostatic urethra following excision of a fibrous obstruction by the cutting high-frequency current; perhaps in that respect he had been fortunate. The character of this current was very different from that of the coagulating current. Beer, who visited his (the speaker's) clinic at New York, saw there a patient operated on three years previously, and said, "In your case the bladder neck stands wider open three years after operation than in my cases in which I open it, and perform a suprapubic cystotomy."

In answer to Mr. T. J. Millin, he had used the arc, but in his hands it had been a tube-electrical machine instead of a spark-gap, and had caused more bleeding at the time of the operation, while the post-operative bleeding had been very serious. He did not think perfection had yet been reached in these electrical machines.

He had had patients who had a cardiac lesion, or renal insufficiency to such a degree that they could not stand a suprapubic operation or a perineal prostatectomy; and to them he had said, "If you like to take a chance I may do you good, or I may not." In such cases he had been able to whittle off pieces and reduce the residual urine, and so render the patients comfortable. But he considered it the surgeon's duty to lay his cards on the table before the patient, rather than promise a good cure by trans-urethral surgery.

Mr. Winsbury-White had mentioned the point of making a correct diagnosis. Bladder necks in America looked the same as those in England, and it was sometimes difficult to make a diagnosis; *then* it was wise in such a case to look with both instruments (urethroscope and cystoscope), and decide on the composite picture.

In answer to Mr. MacDonald, he had had one or two cases in which he had been unable to pass an instrument; in these, by means of the continuous irrigation, he was able to distend the posterior urethra enough to cut from the posterior urethra through the bladder neck. That was the only way in which one could handle that type of case.

Section of Epidemiology and State Medicine.

[*November* 27, 1931.]

William Budd: a Forgotten Epidemiologist.

By E. W. GOODALL, O.B.E., M.D.

WILLIAM BUDD was born in 1811 at North Tawton, a Devonshire village, at that time containing some 1,100 to 1,200 inhabitants, where his father, Samuel Budd, was a general practitioner. His early education was received at home. When he was 16 years of age he was sent to Paris to study at the Collège de France and the École de Médecine. He "commenced his medical studies," as he has himself informed us, "as pupil of the celebrated Broussais, and continued them for a long time under his guidance" [31]. It was in Paris in Louis's wards at La Pitié that he became acquainted with typhoid fever [7]. Among his French teachers there were also Andral and Cruveilhier. In 1836 he was attending the practice of the Middlesex Hospital [31], where he was a pupil of Watson, who formed a high opinion of his ability. During his early professional career he was, for seven years, engaged in a country practice.

Budd graduated as a Doctor of Medicine of the University of Edinburgh in 1838. One of his biographers, Bettany (Dict. Nat. Biog.) states that he studied in that city, but nowhere in his writings does Budd himself give even a hint that he did so. Dr. John D. Comrie has kindly informed me that in those days it was possible in an exceptional case for a man to present himself for examination without previous residence in Edinburgh.

William Budd therefore appears to have received a more thorough medical education than fell to the lot of the medical practitioner of his day. His ability was soon recognized. Not long after he graduated he was appointed Physician to the Dreadnought Hospital, but he occupied that post only for a short time. He was also selected to write the articles on such important subjects as gout and rheumatism for the fifth volume of Tweedie's *System of Practical Medicine*, which was published in 1840.

In 1841 or 1842 Budd settled in Bristol and soon afterwards was appointed Physician to St. Peter's Hospital, at that time a Poor Law Infirmary.

In 1847 he was appointed Physician to the Bristol Royal Infirmary, a post he held till 1862. He was also for ten years Lecturer on the Practice of Physic in the Bristol Medical School. In 1870 he was elected to the Fellowship of the Royal Society. He died on January 9, 1880, at Clevedon, in Somerset, after a painful illness of six and a half years' duration, apparently due to an affection of the brain, for Michell Clarke states that he was afflicted with right hemiplegia (*Brit. Med. Jour.*, 1880, i, 63). He had married a Miss Hilton, by whom he had a large family.

When Budd took his degree at Edinburgh he was awarded a gold medal for a thesis on rheumatism. In 1840 he competed for the Thackeray Prize for the best essay on certain questions concerning continued fever [7]. "The prize was not awarded to him because in the opinion of the judges—of whom Sir John Forbes was umpire—the ideas enunciated in his essay were too novel or startling" (Clarke).[1] From a statement made by Budd in one of his papers on typhoid fever, written several years later, it appears that the object of the essay was to show that typhus and typhoid were two different diseases, that they were both contagious, and that neither was of spontaneous origin. He also states that he read an abstract of his essay before the Bristol Medical Library Society on February 18, 1843. The minutes of this Society are no longer in existence. It is a great pity that Budd's

[1] For an account of this prize see the *British Medical Journal*, 1931, ii, 396. The successful competitor was Dr. W. Davidson, of Glasgow, whose more orthodox essay will be found as an Appendix to Vol. xi, 1841, of the *British and Foreign Medical Review*, of which Forbes was editor.

essay was not published; had it been, he would rank amongst the earliest advocates in this country of the difference between the two diseases, for it was not till 1850 that Sir William Jenner placed the question beyond doubt.

To his strictly professional attainments were added other qualifications which raised him above the common level. He possessed a good knowledge of French, German and Italian, and his writings bear evidence that he had read widely the classical authors, ancient and modern. He was, moreover, an accurate draughtsman and photographer, and made frequent use of his skill to illustrate his papers and addresses. Bubbling over with enthusiasm, too, he must always have been. No one but an out-and-out enthusiast would, in the month of August, make a post-mortem examination on the whole carcase of a pig, dead at least three days from fever with intestinal ulceration, and sent at his own request from Edinburgh to his private house in Bristol. But Budd did so in spite of the fact that there was so much decomposition and such a horrible stink that, as he says, even his pathological zeal was put to a test of no uncommon severity [19]. His energy must have been beyond the normal. Michell Clarke states that he acquired an "immense practice." Yet he contrived to undertake extensive epidemic and epizoötic investigations and to enter actively into the affairs of the British Medical Association, which even in its early days was a very energetic body. The practical turn of his mind is shown by the way he carried or tried to carry his views on contagion into practice, with antiseptics for human beings and the pole-axe for animals, and by his zealous promotion of the Bristol Water Works.

As regards his appearance he is described as having "a strong-looking robust body," and as being "of a good height, well made and of a brave presence." Possessing a vigorous and acute intellect, he was yet highly sensitive and emotional. To judge from his writings he was deeply imbued with a strong sense of duty to his fellow citizens, poor as well as rich.

When we are trying to estimate at their proper value the writings of authors of the past, we have constantly to guard ourselves from unconsciously interpreting their conclusions and opinions as if they had reached them with the aid of the knowledge we possess and they did not; otherwise we may be led seriously to undervalue them, or lightly to dismiss them as superfluous. We must, therefore, endeavour to place ourselves in their surroundings and to limit our thoughts to the circumference of their knowledge. I have been at some pains to ascertain what was known and, what is quite as important, what was believed, about epidemiological subjects by William Budd's contemporaries. Budd's activities may be said to have extended over a period of thirty-five years, from 1838, the year of his graduation, to 1873, the year when he was completely laid aside by his long and fatal illness. During those years the mind of the medical profession was much exercised on three important epidemiological questions: (1) the nature of contagion; (2) spontaneous generation; and (3) the causes of endemic and epidemic diseases, especially the latter. There were several subsidiary points, to which I may have to refer, but those were the three chief, I may even say disturbing, topics. I will as briefly as I can put before you the views which were then held on those subjects, and I shall confine myself almost entirely to British opinion, to convert which Budd's efforts were in the first place directed.

And first, as to contagion. The fact of contagion was admitted by the majority, though there were a few who denied its existence in any shape or form, such as Dr. John Parkin and Miss Florence Nightingale, whose voice was so authoritative with the public at large. Then there were those who, while admitting its existence, held that its agent was an inanimate poison of the nature of a ferment, produced by some morbid change in the blood of the infected person, and passed on to a healthy person in whom it set up a similar change. This hypothesis is still perpetuated in the term zymotic, first applied to the group of infectious diseases by Dr. William

Farr. This view was held by many eminent men, most prominent amongst whom I may mention Sir Thomas Watson. Sir B. W. Richardson believed the agent to be an animal alkaloid. Then there were those who were agnostic or indifferent on the subject. Recognizing the fact of contagion, they either kept an open mind as to its nature or did not trouble themselves about it in any way. Judging by their writings in textbooks some of the most distinguished men adopted this attitude, such as Sir Robert Christison, Dr. Bright and Dr. T. Addison. Lastly, there were those who had revived the doctrine that the agent of contagion was living, and amongt these, too, there was diversity of opinion concerning its real nature. While one party, as conspicuous in which I may mention Dr. Edward Lancaster and Dr. Lionel Beale, was of the opinion that the agent was an altered leucocyte or some particle of living and perhaps degenerate bioplasm in an animal or human being, derived from similar leucocytes or similar particles of bioplasm, another held to the view that it was something quite foreign to human or animal bodies, which it invaded and in which it multiplied. Amongst the early supporters of this view were Sir Henry Holland and William Budd. The foundations of the modern germ theory of communicable diseases were laid in Budd's lifetime, but he did not live to see the completion of the edifice. Even in the early days of bacteriology there were those who regarded the newly-discovered micro-organisms as the products of disease or as secondary invaders.

I alluded just now to the revivalists of the doctrine of the living agent of infection. I need hardly say in this Section that the doctrine was a revival of that put forward by Jerome Fracastor three hundred years earlier. It is singular, however, how little the revivalists recognized or admitted that they were revivalists. In their writings I find no reference to any authority earlier than Kircher (1658), and usually there is no reference at all to previous writers. The history of Fracastor's conception by no means redounds to the credit of his successors up to the time of the revival. He taught the doctrine of the *contagium vivum* very clearly, and his method of treatment, preventive as well as remedial, of the contagious fevers was based upon it. The doctrine was neglected and forgotten, but in some parts of Europe his methods of treatment were to a certain extent kept alive, possibly by purely commercial interests.

The real revival of the doctrine of the *contagium vivum* dates from the discovery by Cagniard-Latour and Schwann, independently in 1836 and 1837, of the mode of propagation of yeast-cells and their rôle in fermentation; but nearly fifty years were to elapse before Pasteur and his disciples established the germ-theory of disease. Meanwhile Dr. (afterwards Sir Henry) Holland, in 1839, and Professor Jacob Henle, of Berlin, in 1840, had revived the doctrine, relying for its exposition mostly upon reasoning by analogy from certain occurrences they and others had observed in the world of nature. Budd refers to the writings both of Holland and Henle. Holland's views on the subject are to be found in the book entitled *Medical Notes and Reflections*, and in the second and third editions of this work he quotes Henle, by whose writings he was evidently influenced. Budd was certainly a supporter of the view that the agent of contagion was living, so that he must be reckoned amongst the revivalists of the days before bacteriology, which can hardly be said to have attracted much notice before Pasteur's work on fermentation in 1857.

A subsidiary point in the controversies of those days was that of the specificity of the various agents of infection, whether they were regarded as animate or inanimate. Specificity had been admitted by Marten when writing of consumption in 1720. But there were those who held that all the infections were only modifications of a single, universal contagion, as Bosquillon, the French translator and commentator of Cullen, puts it. This view was thought worthy of attention by Dr. William Addison, F.R.S., of Maidstone, as late as 1854.

As regards spontaneous generation it may be a matter of surprise to some of us to learn that less than a century ago this doctrine, so far as the origin of certain,

if not all, of the infectious diseases was concerned had a firm hold on the mind of the medical profession. Our surprise, however, will be lessened when we remember that at that time the large majority did not believe in the *contagium vivum*, and for them it was not difficult to imagine that amidst the highly insanitary conditions in which the people—and not only the poorest of them—lived, poisonous, chemical substances, gaseous or solid, might readily be and frequently were evolved from decomposing organic matter, to cause disease in men and animals. Still, the acknowledgment of a *contagium vivum* is far from being incompatible with that of spontaneous generation, a doctrine which Fracastor held and Sir Henry Holland did not entirely reject. It is a fatalistic and therefore a comforting doctrine to the believer. It saves much thought; also much action, especially action which may interfere with business interests. When cholera first invaded this country at Sunderland in 1831, the suggestion that it had been introduced from without was strenuously resisted by medical men and laymen alike; granting that the disease was true Asiatic cholera, it was bred locally; to admit its foreign origin meant submission to quarantine regulations, irksome to trade. And if, as Budd pointed out more than thirty years later, spontaneous generation really took place it was "a waste of power to be taking precautions against the importation from without of what might turn up any day as a home product." Still, even some of those who acquiesced in the animate nature of the agent of infection, thought that it might on occasion arise of itself. The doctrine died hard. As late even as 1907 Dr. Charlton Bastian read a paper in which he advocated it before the Royal Medico-Chirurgical Society. The principal stumbling-block to the rejection of the doctrine was the sporadic case, and especially the one which failed to originate an outbreak of disease.

At the time of which I am speaking no real advance had been made on the views concerning the causes of endemics and epidemics that had been held for centuries, and miasms, indefinite vaporous mysteries, epidemiological wills-o'-the-wisp, supposed to be generated in decomposing animal or vegetable matter, or released from the soil by the action of earthquakes or volcanoes, were deemed sufficient to account for the occurrence of epidemics. After some of the facts of electricity had been brought to light, that force was invoked as an auxiliary, if not as the principal, agent. Of Sydenham's doctrine of the "epidemic constitution" I find little mention in the literature of the time. It was rejected by both Holland and Budd.

In most of these views contagion did not play an important part; indeed, there were those who, while convinced of the fact of contagion, denied that it had anything to do with epidemics, and separated contagious diseases as a class from epidemic diseases. This question was discussed as late as 1861 by Dr. Gavin Milroy, at a meeting of the Epidemiological Society. He came to no definite conclusion. Others were of the opinion that a miasm could start an epidemic, but that there was a something which might and did come into play to intensify the propagation of the disease, and that something they held to be contagion. This idea can be traced back to Fracastor. In later times it was known as the doctrine of the "contingent contagion."

From this brief account I think it is clear that William Budd appeared on the scene at a time when the views of the medical profession on epidemiological subjects generally were in a most confused and nebulous state. Although he began to publish papers very soon after he took his degree, he did not put into print anything on infectious diseases till September, 1849, when his pamphlet on malignant cholera appeared. But that he had been thinking much on the subject is evident from the fact that in that paper he comes out with very clear, concise, and on the whole correct views on the origin, spread and cessation of a cholera epidemic. He had touched on the question of contagion in a paper on cancer in 1842, in which he discussed the communicability of that disease. We remember, too, that in 1840

he had entered an essay in the competition for the Thackeray prize on the subject of continued fever.

Budd wrote more often and at greater length on infectious than on other diseases. His papers on the latter it is not in my province to discuss. They afford good evidence that he was a careful and accurate observer and that he took and preserved notes of his cases. His *Retrospect of Anatomy and Physiology for the year 1843-44* is a most comprehensive survey and contains much discerning criticism. In it he deplores the apathy of the State in respect of the advancement of science, and castigates certain French physiologists for the performance of what he considers to be quite unnecessary experiments on animals, in terms which are reminiscent of the censure passed by Celsus on Herophilus and Erasistratus for their inhuman practices.

I have appended to this paper a list of Budd's published writings, from which it will be seen that the infectious diseases he deals with are cholera and typhoid fever, those two especially; then malignant pustule, sheep-smallpox, rinderpest and typhoid fever of the pig; and, lastly, phthisis and scarlet fever; while his papers on diseases other than the infectious, with the exception of the *Retrospect* [33], are devoted to elucidating special clinical, pathological and therapeutic points, those on the infections are concerned mostly with matters of general interest common to them all—wide, epidemiological questions in fact.

It is clear from his writings that from the very beginning Budd vigorously attacked the prevailing notions on the nature and importance of contagion. I will take in order the three burning questions which I referred to as agitating the profession in his time, and show how he dealt with them. To us the real interest lies in observing how the question of the *contagium vivum*, and all that depended upon its solution, was approached in the days before the evidence brought forward by the bacteriologists was available. First, there was the nature of contagion. Budd was with the majority in being a contagionist, but with a minority in holding that the agent of contagion was a living organism. In his very first paper, that on malignant cholera, dated September 27, 1849, he begins a series of dogmatic statements with the bold assertion "that the cause of cholera is a living organism of distinct species." This, with the other statements, is according to him a deduction from facts he has observed.

In the days immediately before the era of bacteriology those who argued in favour of a *contagium vivum* were better off for evidence than was Fracastor, in that they had the aid of the microscope and such discoveries as had been made with it up to that time. But that instrument had been of use in this particular question only in so far as it had shown that there were in existence minute living organisms too small to be seen with the naked eye. As a matter of fact, although we now know that Budd's statement, as it stands by itself, is perfectly true, he had at that date no solid grounds for making it. Budd came sadly to grief over this supposed cholera organism, the discovery of which was the occasion of his paper. Certain members of the staff of the Bristol Royal Infirmary, Budd amongst them, had been examining with a microscope the stools of cholera patients and the water and air of infected places, and they had observed certain objects which they believed to be fungi. Budd gives drawings of them in his paper. The fate of these bodies reminds me of that of the "fossil bones" from which Mr. Brown, of Calaveras, reconstructed "an animal which was extremely rare," as told in Bret Harte's amusing ballad; for about three weeks later certain medical men, by name William Baly, William W. Gull, William Jenner and John Marshall, were unkind enough to publish a paper in which they proved that these bodies were elements of partly digested food, calcareous substances derived from medicines, especially chalk mixture, fat globules and starch cells. Budd learnt a lesson he never forgot; never again did he speak so positively concerning particular micro-organisms. In 1865 he translated an extract from a French article on

Davaine's discovery of the *Bacillus anthracis*. In commenting upon it he expresses the opinion that further data are required before the case for that organism is established, and referring to the discoveries of various organisms in disease, writes as follows: "In all such cases there is a special danger, which those who most study the subject will best appreciate, of falling into the old error of taking for essential what may be an epiphenomenon." Even in his last published work, the book on typhoid fever, 1873, he expresses himself on the subject with great caution.

Budd relied for his firm belief in a *contagium vivum* on those arguments which alone were available in pre-bacteriological days: the fact of the incubation period in infectious diseases, the manner in which those diseases spread, the phenomena of inoculation in such diseases as smallpox and the protection afforded by one attack against another.

On the question of specificity he was on safer ground. The fact that the infectious diseases "breed true" and that one of them does not protect against another were the chief arguments in its favour.

Against the doctrine of spontaneous generation, whether in general, or as regards typhoid fever in particular, he waged an incessant and uncompromising war. I do not think it necessary at this time of day to set forth the arguments against it. With the argument drawn from the sporadic case combined with that which insisted that, as the first case of an infectious disease must have arisen spontaneously, so fresh cases may be generated in a similar way, an argument which even so illustrious a physician as Trousseau had recourse to in respect of typhoid fever, he employed sarcasm with great effect: "Let an isolated case of typhoid fever occur, and, merely because he cannot trace the disorder to its source, many a medical man will assert, without the slightest hesitation or doubt, that the disease has, clearly, originated spontaneously. Press him and he will be ready at once with the argument that there is no reason why the conditions which *first* gave rise to it may not again give rise to it now. But let a young sinner with the peccant mark upon him swear ever so stoutly that the disease he bears has come on spontaneously; let him argue as plausibly and as long as he will against the incredulity which refuses to see that the conditions which *first* gave rise to this disease may again give rise to it now, and the very same medical man will laugh him to scorn."[1] Curiously enough, however, only a few months earlier, Dr. Milroy, in the paper to which I have already referred, informed the members of the Epidemiological Society that "it seems more than probable that the gonorrhœal poison may be generated *de novo*, as cases are continually occurring in practice without any reasonable suspicion of antecedent contagion." He also was of the opinion that syphilis might arise in the same way.

To the alleged spontaneous origin of typhoid fever Budd paid special attention because no less an authority on that disease than Murchison strongly advocated it. Indeed, Murchison had proposed and made use of the name "pythogenic" instead of "typhoid" fever, for the reason that, in his opinion, the fever mostly arose from putrescent matter, one of those question-begging designations which are so serious a hindrance to a rational nomenclature. Murchison was no believer in the *contagium vivum*, though he recognized contagion. In the case of typhoid fever the contagion was generated in putrid and decomposing organic matter, including the stools from a typhoid patient some time after they had been passed from the bowel, for fresh stools were not infectious. The contagion was never developed in the human body. Therefore when a case of typhoid arose it was not necessary to look to a previous case for its origin. Budd, of course, would have none of that. Murchison devotes twenty-seven pages of his book on continued fevers to the question of the independent origin of typhoid, but he allots only a short paragraph of nine lines to answer the cogent argument that solitary cases of such an admittedly contagious

1 *Brit. Med. Journ.*, 1861, ii, 625.

disease as smallpox are by no means unknown and yet are not usually attributed to spontaneous generation All he has to say in reply is that such cases are very rare, and that the poison of smallpox is very virulent. He had apparently forgotten Dr. Gregory's well-known statement that "of the numerous cases received into the smallpox hospital not one in twenty is ever able to trace the disease to any source of infection." Murchison also completely ignores Budd's evidence as to cases of typhoid which, being concealed either ignorantly or wilfully, may easily be the origin of supposed spontaneous cases.

In my opinion it is when he is discussing general epidemiological questions that Budd is seen at his best. The exact nature of the contagious agent is a matter of experimental investigation, and in the days before bacteriology neither he nor anyone else could be absolutely certain that the agent was a living organism. But, the fact of contagion being admitted, as it was by most of his contemporaries, a good deal could be deduced towards the explanation of epidemics, and still more if it was supposed that the agent was living. I have mentioned the views which were generally held during Budd's lifetime. The miasmatic doctrine held the field; the rôle played by contagion was subordinate; indeed there were those who rejected it altogether from the cast as a useless supernumerary. For Budd, however, contagion was a factor of the first importance; "a great cardinal truth," "a fundamental proposition," "the MASTER-fact which is the key to all the rest," are phrases which he uses. For the evidence in its favour he relies chiefly upon his personal experience of epidemics and outbreaks of cholera in Bristol and elsewhere, of typhoid in North Tawton and other places, of sheep-pox in Wiltshire. The manner in which these epidemics progressed is not that of a miasm. Writing in 1849 of cholera he says: "Wherever the disease is introduced into a new community it takes time to develop itself. First a few dropping cases occur with more or less irregularity and then gradually multiply, but it is generally some months yet before the disease bursts out in its full virulence. Plainly this is not a pestilence caused by a miasm diffused widely through the air and producing its full effect at once, but one that grows as it goes and spreads by the gradual increase of the peculiar agent which is the instrument of its deadly work." And again, in 1854, after relating some striking instances in which the infection of cholera had been carried from Plymouth by persons infected in that town to uninfected villages several miles away, he writes: "Speaking plainly then, the case stands thus: either facts such as those related prove cholera to be a catching or contagious disorder, or no facts can prove it to be so; or, to enlarge the proposition still further, no facts (short of inoculation) can prove any disorder to be contagious, which is a *reductio ad absurdum*. Try as you may you cannot escape from the dilemma." Generally, he argues from the facts of the epidemics he witnessed: why should a miasm attack only those places (countries, towns, villages, houses) into which a case of the disease has been introduced, and leave untouched other places quite close to them and similarly conditioned, but lacking the introduction of the disease? In all his writings he impresses upon his readers the importance of contagion as a factor in the spread of an epidemic. It must be remembered that Budd was a very practical man, and his persistent insistence on this point was due not so much to the pleasure of making an academic demonstration as to the conviction that by a recognition of the fact an epidemic could be cut short or prevented by adopting appropriate measures.

I will here permit myself to make a few remarks upon Budd's insistence upon the importance of contagion in two diseases in particular, typhoid fever and phthisis. It is chiefly in connection with the contagiousness of typhoid that Budd's name has survived in medical literature, and, as regards that, in connection with the North Tawton epidemic. This epidemic began with a single case in July, 1839, and in the course of a few months not only spread widely in that village but invaded three

neighbouring places. In respect of this epidemic Budd has been criticized by two eminent medical men, Murchison and Creighton, the one a practising physician of wide experience, especially of the infectious diseases, the other a learned historian of extensive reading, but whose knowledge of the guises in which diseases present themselves was derived almost entirely from books. I will deal with his criticisms first.

Creighton doubts Budd's diagnosis of the cases in the North Tawton epidemic and believes that he mistook typhus for typhoid, and he complains that Budd gave insufficient clinical evidence in his account of the epidemic. I have no hesitation, however, in stating that Budd's diagnosis was undoubtedly correct. There is quite sufficient clinical evidence in his account to recognize with certainty the nature of the disease. Moreover, Budd was perfectly well acquainted with the difference between typhus and typhoid. He discussed that point in his essay for the Thackeray Prize, and it is almost certain that he was engaged in the preparation of that essay at the very time of the North Tawton epidemic. He also saw much typhus in Bristol, to which city it was brought from Ireland. Typhoid fever was much more common in England in 1839 than Creighton was prepared to admit, and he did not believe in its contagiousness. *Hinc illæ querelæ.* But Creighton justly convicts Budd of error when the latter "treats the epidemic of 1838-39 all over Britain as purely one of typhoid." Budd ought to have remembered that a large proportion of the cases must have been typhus.

Murchison attacked Budd on the question of contagion. He did not doubt the diagnosis, though if he could have shown that the disease was typhus there was an end to Budd's contention so far as that particular epidemic was concerned. Murchison's position in regard to the communicability of typhoid is rather difficult to understand, because of his inconsistency. Thus on page 489 of the second edition (1873) of his work on the continued fevers, writing of the North Tawton epidemic, he endeavours to weaken Budd's evidence by stating, quite correctly, that "although the date and locality of the first case are mentioned, it is not stated that the patient caught the disease away from the place." He has evidently forgotten that on page 464 he has written of the same epidemic as follows: "During the prevalence of the fever, it so happened that three persons left the place after they had become infected, and all three communicated the disease to one or more of the persons by whom they were surrounded in the new neighbourhoods to which they had removed, although in each of the three localities there had been no cases of fever previous to their arrival."

The North Tawton outbreak has not infrequently been appealed to by writers since the days of Budd and Murchison as evidence of the direct transference of the infection of typhoid fever either by the soiling of the hands by contact or through the air for a very short distance, a few feet or inches. As a matter of fact, however, Budd nowhere states in what way he believed the infection was spread in that epidemic. He was concerning himself solely with the master-fact that the disease was communicable, and not with the exact method of its transmission. In this connection it is well to call to mind what Budd meant by the word "contagious." He states more than once in his writings that he uses it "in its widest sense, as signifying the communication to others, through whatever medium, of the specific fever which is the subject of inquiry, by persons already infected with it." We nowadays prefer to employ the word "infectious" where Budd and his contemporaries (including Murchison) used the word "contagious." Budd's definition, borrowed word for word from Piedvache, is, perhaps, somewhat loose, but it is clear what he means. Contagion is an action by which an infecting agent is conveyed from the sick to the healthy by various means through various channels. This definition dates back to Fracastor, who, you will remember, has a chapter on contagion which is effected at a distance—a distance, that is, between the infecting and the infected person.

There is not the slightest doubt that Budd did think that the infection of typhoid was in many cases acquired by actual contact or through a short aerial space, for though nowhere in his account of the epidemic does he say a word about the water supply of North Tawton, in a later paper he states that those of the neighbouring villages of Chaffcombe and Loosebeare, to which the infection was carried, were beyond suspicion.[1]

Budd's *Memorandum on the Nature and Mode of Propagation of Phthisis* is worthy of being recalled to memory, not only because of its intrinsic merit, but also because of the circumstances of its conception, birth and reception which, I venture to say, constitute one of the most curious, but almost forgotten[2] incidents in the history of British medicine. It is dated December 6, 1866, but it appeared in the *Lancet* of October 12, 1867. The thesis, which it is the object of the memorandum to set forth, is that phthisis is a disease which does not originate spontaneously, but is communicated from one person to another. Budd writes that the idea came into his mind quite suddenly, "unbidden, so to speak," while he was out walking in August, 1856. It was suggested to him by what he believed to be the close analogy between typhoid and phthisis. It is quite clear from what he states that he believed he had established and was proclaiming a new fact; and that, in his opinion, it was also an important one is evident from his action; for, perhaps fearing, like Galileo with his famous cryptogram about the planet Saturn, that he would be forestalled in the merit of his discovery, he enclosed the memorandum in a sealed packet and sent it to his friend, Dr. George Paget, at Cambridge, with the request that the packet should be opened and its contents made public only when the sender so directed. The memorandum was an epitome of a reasoned and detailed paper which it was his intention to prepare and publish in the course of a few months. Finding, however, that he was unable, on account of the engrossments of professional practice and other circumstances beyond his control, to carry out this intention, and very likely also, it seems to me, because he had become aware of Villemin's experiments on the transmission of tuberculous matter, an account of which had appeared in the *Lancet* of March 9, 1867, and further, of Italian opinion on the subject, he gave directions in the September of the same year for the unsealing of the packet. Dr. Paget read the memorandum and sent it to the *Lancet*.

Budd, as I have said, believed his views to be original and new. So did Dr. Paget, for he wrote to that effect in a letter to the Editor of the *Lancet*, which prefaced the memorandum. So did Dr. Cotton, senior physician to the Hospital for Consumption at Brompton. So also did Dr. Samuel Wilks, physician to Guy's Hospital. In the *Lancet* for November 2 there appeared a paper by Dr. Cotton, in which he states that he does not at all agree with Dr. Budd's views and that in his opinion phthisis is a "purely constitutional disease, which may be inherited or acquired, but is incapable of being communicated from one person to another *in the ordinary sense of a contagious disease*." He admits that "it is not impossible under certain circumstances . . . . this constitutional condition may be conveyed from husband to wife, and, perhaps, but with less probability, *vice versâ*." A week later, in the same periodical, we find Dr. Wilks writing as follows: "As if at the present day we had learned nothing and it were still an open question whether tuberculosis was a diathetic disease or a mere accidental affair like small-pox, dependent on risk of contagion. I cannot consider this an open question." He supports the views of Dr. Cotton and states that he has "no doubt that similar views are held by most of the teachers in our schools." He regards Dr. Budd's memorandum as very "remarkable" and his theory as "novel." A statement in

1 He tells us, however, that since the epidemic a supply of pure water has been provided at North Tawton; from which statement we may infer that the water had previously been impure.

2 The conception and birth are related by Sir James K. Fowler in *The Sthenics*, 1930.

the *Lancet*, to be quoted later, also shows what the general opinion of the profession on the subject was at that time in this country.

Now what appears to me to be remarkable in all this is that we find four of the leading physicians of the day—Dr. Budd, who had been lecturer on physic at the Bristol medical school, Dr. Cotton, who as senior physician to the Brompton Hospital may be regarded as *a*, if not *the*, leading authority on phthisis of his day, and two well-known clinical teachers in prominent medical schools, both of whom attained high professional reputation and honours, one and all evidently firmly convinced that the idea that phthisis is contagious was in their day novel. They might be forgiven if they had regarded it as a disproved or discredited notion of the ancients, as possibly Dr. Wilks did; but, as a matter of fact, perhaps through the continuance of Fracastor's teaching three hundred years earlier, a teaching which was quite clear and even emphatic, though by no means novel even in his time, the belief in the contagiousness of phthisis had persisted in no uncertain guise and had been the cause of various sanitary regulations in Italy, Spain, Portugal and parts of France right down to the very period of which I am now speaking. And, to bring the fact right up to the date of Budd's memorandum, there actually appeared in the *Lancet* for March 9, 1867, under the heading "Medical Annotations," the following paragraph: "It seems that the Italians have conveniently imbibed the notion that consumption is contagious, and should unhappily a phthisical patient die in the hotel, the bill but too often contains the singular item: 'Indemnité pour réfaction des meubles et de la chambre occupée par le défunt, £100 sterling.' The ground upon which such a charge, the most exorbitant of any that has been manufactured for many a long day, rests, is utterly untenable and, of course, the demand should be at once repudiated."

The notion that phthisis is communicable does not appear ever to have been held generally or strongly in this country until a few years ago. Towards the close of the seventeenth century Richard Morton in his book *Phthisiologia* (English translation, 1694) gives infection as one of eleven causes of "an original consumption of the lungs," writes that he has observed by frequent experience that it does, like a contagious fever, infect those that lie with a sick person, and relates the histories of two persons who, he believed, caught the disease in that way. In 1720 appeared Benjamin Marten's *A New Theory of Consumptions*, to which I have already had occasion to allude. In this book the author writes that the "prime or essential cause of consumptions" is possibly to be found in certain species of animalcula or their ova, such as had been observed by Leeuwenhoek and others with the microscope, which are able to pass in various ways from the sick to the healthy. But neither Morton's experience nor Marten's theory appears to have had weight with their professional brethren, and the reference to the subject in *Humphry Clinker* can be taken as evidence of British ignorance. Apparently Squire Bramble was the only one of the frequenters of the public baths at Bath who had any inkling of the contagiousness of consumption; but he had made the grand tour in his youth, and Smollett had resided in Italy for some time before he wrote his entertaining novel, which was published in 1771. The admission of the possibility of the infectiousness of phthisis by the venerable William Heberden, through the piety of his son,[1] in 1802, and by the erudite and versatile Dr. Thomas Young in 1815, seems to have been of no avail in reviving the belief, so that by Budd's time all knowledge of it appears to have died out, just as had that of the *contagium vivum*; and in spite of Koch's discovery of the tubercle bacillus in 1882 many years were to elapse after the publication of Budd's memorandum before the doctrine he advocated was fully recognized and acted upon in this country.

As stated above the memorandum gives only an epitome of the evidence Budd intended to produce in support of his conclusions. Apart from the

[1] William Heberden, the elder, the author of the *Commentaries*, died in 1801 in his ninety-first year.

demonstration of the tubercle bacillus, the evidence derived from experiments on animals, and what we now know of the convection of the bacillus in milk, his evidence is just that which would be brought forward at the present time as corroborative of those basic facts. It is much to be deplored that Budd never produced a reasoned and detailed paper as he intended. Had he done so, possibly more notice would have been taken of his conclusions.

There are two papers by Budd which, in my opinion, would establish his claim to recognition as an epidemiologist in the fullest sense of the word, even if he had published nothing else. They are the paper on *Variola Ovina* (*Sheep's Smallpox*), and the memorandum on the *Investigation of Epidemic and Epizoötic Diseases.* The former constituted the address on medicine which he delivered at the annual meeting of the British Medical Association at Bristol in 1863. Of it Michell Clarke wrote that for more than an hour Budd held an overflowing audience entranced. In this address, which I venture to say will repay perusal even now, Budd sets forth the results of a piece of field-work he had carried out, apparently quite voluntarily, in the case of the severe epizoötic of sheep-pox which broke out at Allington in Wiltshire in June, 1862. Starting from a single case the disease spread in the course of a few months to eight other farms, extending over many square miles, and before the outbreak ceased eight to ten thousand animals had been infected and hundreds of them had either died naturally or been killed.

In this paper Budd not only discusses the questions of the nature of the infectious agent and of spontaneous generation, which had been alleged as the origin of the outbreak, but he takes up much wider epidemiological problems, which, so far as I have been able to discover, had never before been seriously considered. He points out why it is that so little is known of epidemiological laws. It " may be mainly traced to two causes; first, to an exaggerated estimate of the difficulties of the subject, and second, to want of clear views as to the method to be followed in its investigation. Authors have occupied themselves far too much in dwelling on the inscrutable nature of epidemic poisons, and in accumulating epithets to signify the hopelessness of attempts to discover the laws of their action. This practice hinders the advance of science, by discouraging inquiry, and still more fatally by begetting a prejudice against precise views, on the plea that such views are unattainable, and hence cannot be in accordance with the truth." Not that he is unaware of the difficulties of the subject—far from it; " the material causes of epidemics are invisible; and the laws which govern invisible things must needs be hard to make out. But that they may be perfectly made out is clear from this, that the invisible powers of nature, such as gravitation, for instance, are the very powers of which our knowledge is the most sure and precise." However, " the problem of the epidemiologist has in it at least one element of simplicity, . . . its chief concern is with the history of a single cause . . . . the biography . . . of a single species." He is here referring to the factor of the specific infection. From this and other passages in his writings it might be inferred that Budd was one of those who believed that once the existence of living contagia was granted the explanation of epidemics was simple. But other passages can be given which show that he was quite aware that the *contagium vivum* was not the only factor in the problem. I shall quote one such passage presently. Budd then goes on to make some observations on what he states was the method most in vogue in epidemiological inquiries, namely, the accumulation of " a mass of statistical details—meteorological, physiological, topographical, and other—relating to the particular epidemic disease which may happen to be in question, often with the most impartial indifference as to whether these details establish anything of importance or not. . . . Evidence so collected is no doubt of value, but chiefly as furnishing data for the real discoverer to work upon. Statistics afford much information on epidemics, but it is all of a general kind. The really vital questions they leave

almost untouched. Neither on the mode of being of the morbific agent without the body, nor on its mode of action within, do they throw any but a dim and distant light." The mass of statistical details to which Budd alludes was doubtless the various tables published in the annual reports of the Registrar-General. Budd makes very little use of statistics in his arguments. The most he does is to quote a few figures, in some instances erroneously. The problems he was trying to solve, and especially those of the nature of contagion and its relation to epidemics, were not to be unravelled by statistical methods. Even to-day, with a much larger and more trustworthy volume of statistical data at his disposal than was available in Budd's time, one of our highest authorities on these subjects candidly admits "that the statistical method is limited and selective; that it selects some only of the possible objects of study, and these, perhaps, not the most important . . . there are many more things in heaven and earth than are recorded in the annual reports of the Registrar-General, or are amenable to the calculus of correlation." I am quoting from an address given only a few weeks ago by our President at the annual meeting of the British Medical Association. Curiously enough, too, Budd in his address, delivered sixty-eight years ago, also before a meeting of the Association, anticipates Professor Greenwood and the school of experimental epidemiologists on two other points, when he urges the necessity of limiting the scope of epidemiological inquiry, if any notable advance in knowledge is to be made, and indicates how limitation is to be ensured. "We must," says our President, "by a deliberate limitation of the field of observation, try to discover which of the vast number of possibly influential factors are likely to repay study in the world of 'real' epidemics. Within that limited field we must use the problems of statistical description which, although they have not enabled us to solve the problems of epidemicity, have at least been valuable instruments of sanitary administration. That is the case for experimental epidemiology." And he goes on to explain why and how the experiments are carried on in herds of mice. Now listen to Budd: "The first position I would take, therefore, is that if we wish to obtain clear views on the subject of epidemics generally, our surest plan is to begin with the study of the contagious group. The next position speaks for itself. It is, that of this group we should select those which, from peculiarities in the mode of evolution of the virus, admit of being propagated artificially, which offer, in other words, the incomparable advantage of being studied experimentally. . . . If we were asked to define what other conditions we would most desire in order to make our task the more easy, our answer would be that all the sufferers should be under our own eye; that their incomings and outgoings and intercourse with one another should be known to us; or, better still, should be determined by our own will. It will be seen at once that, taken together, these conditions are not to be met with in human life. They are only to be found in fact in the case of the domestic animals." The advisability of limiting the field of inquiry (though not exactly in the direction pointed out by Professor Greenwood) and the advantage of using animals as our objects of study are clearly set forth. Those were the reasons which led Budd to investigate the severe, but at the same time localized epizoötic amongst the Wiltshire flocks in 1862, and to pay, subsequently, such close attention to outbreaks of cattle plague and typhoid fever of the pig.

Of course, Budd's immediate object was different from that of Professors Greenwood and Topley, and other workers on these experimental lines. He was only at the beginning of the period of exactness in epidemiological investigation, and was seeking to establish certain general principles which to us appear elementary, but were in his days misunderstood and even disputed.

Budd makes use of this epidemic among sheep in an endeavour to confute the doctrine of "contingent contagion," which was widely accepted at that time. It was noticed then, as it is noticed now, that frequently small localized outbreaks

or even solitary cases of the common infectious diseases occur without an epidemic arising. Sometimes, however, the disorder spreads and a more or less extensive epidemic is the result. There were two varieties of the doctrine to explain this phenomenon. In the one the disease was supposed to be non-contagious at first, and the element of contagion was added in some unexplained way. In the other, while the disease was admitted to be contagious from the very first case, there was added to its quality of contagion, also in some unexplained way, a new quality which enhanced its power of propagation. The solitary case or the very limited outbreak of an infectious disease puzzled the men of a century ago just as it had puzzled Sydenham and still puzzles us. Hence arose the conception of the contingent contagion. Budd pointed out that if nothing more was meant by the somewhat vague phraseology in which the doctrine was presented than that there must be a concurrence of conditions favourable to the propagation of the infection, no exception could be taken to the doctrine: but that was not what was meant and believed.

In my opinion Budd is by no means happy in his endeavour to confute this doctrine. His chief argument is that as sheep-pox had been absent from Wiltshire for about one thousand years, therefore, in looking for an explanation of the epizoötic you can exclude the intervention of any other agency than that of the *contagium vivum* of sheep-pox with its power of reproduction. He appeals to the phenomenon of a swarm of locusts invading and devastating the vegetation of half a continent. When he says that "we all know that there must have been an unusual occurrence of conditions favourable to the multiplication of the insect," he is perfectly right; but he is quite wrong, and is contradicting himself, when he goes on to state that "we all know, with the same certainty, that it is the breeding power of the locust itself that has *alone* [the italics are mine] brought these countless hosts of winged insects into the field." Budd's arguments were good against those who believed in spontaneous generation and against the anticontagionists, but they were not good against the contingent contagionists. He failed to recognize that it was necessary for him to show why it is that from quite a small swarm of locusts countless hosts do sometimes, but not always, emerge. In failing to recognize this he also failed to recognize that the doctrine of the contingent contagion embodies the crux of epidemiology; for though it may have lost its name, it is still with us to-day, in spite of what we know of micro-organisms and viruses. The existence of a contagious agent, living or not, being granted, the conditions by which its dissemination is governed have to be ascertained, and that, I take it, is the principal aim of the epidemiologist.

Another question which Budd deals with in this address on sheep-pox is that of the means by which infection is spread. Before the middle of the last century little, if any, work had been done on the subject since Fracastor had put forth his generalizations in 1546. It was held generally that in most cases the virus was received and given off through the skin, or by way of the air-passages during respiration. The earliest statement I have met with (though I confess that I have not made an extensive search into the question), in which there is any suggestion of another channel, is that made by Nyander, who wrote in 1757 of dysentery, that no person skilled in medicine doubted that the disease was propagated through privies and common sewers, and propounded the far-fetched idea, unsupported by any valid evidence, that the infection is conveyed by means of acari in the dysenteric patient's stools. A few years later, in 1764, Sir John Pringle recorded his opinion that the source of infection of dysentery is to be found in privies, chiefly after they have received the dysenteric excrements, and attributes the spread of that disease in camps and hospitals to straw, bedding and clothing contaminated by infectious excreta.[1] Budd refers with approval to Pringle's views. Mr., afterwards

[1] Both Pringle and Holland look with some favour on Nyander's acarus hypothesis; perhaps because Nyander's thesis was published under the auspices of the great name of Linnæus.

Sir, Patrick McGregor in 1811 suspected that the infection of purulent ophthalmia was conveyed by flies. Henle quotes Hertig as stating in 1834 that a febrile exanthem could be caused by drinking the milk of cows suffering from foot-and-mouth disease. But it was not till after the middle of the nineteenth century that the fact that infection could be conveyed by food and drink came to be generally accepted. Even six years after the publication in 1849 of the conclusions of Snow and Budd on the propagation of cholera, so enlightened a physician as Sir Henry Holland is of the opinion that the view that certain physicians have put forward that the virus of that disease may be conveyed by drinking water is "too doubtful to be admitted as an argument."

Budd was the first to show that the infection of typhoid fever could be conveyed by water, and he was nearly the first to demonstrate the same fact in respect of cholera, in which he was anticipated by Dr. John Snow by only four weeks. I think that there can be little doubt that Snow and Budd independently arrived at this conclusion, but the latter always allowed the former the priority of the discovery. In respect of typhoid, however, Budd showed, first, that drinking water must be contaminated with sewage, and, secondly, that the sewage must contain the specific infection. He also insisted that the infection of typhoid and cholera was conveyed chiefly by the stools, that is the discharges from the organs which are primarily and essentially affected.[1] For the same reason he believed that in scarlet fever the infection lay mostly in the exudations and discharges of the throat and nose. Because in cholera and typhoid fever the infection is cast off chiefly from the bowel, it is to the condition of privies, cesspools, drains and sewers that we must look for an explanation of the spread of those diseases, and he was constantly insisting upon the importance of providing pure water supplies and proper methods of disposing of sewage. He was the more insistent upon these matters because he believed that infection could be given off from sewage to the air. "To inhale, for an instant," he writes, "the exhalations of a sewer, to walk down an infected lane or alley, . . . to drink a draught of water, . . . is often to be stricken by this fever unto death." To us this statement may savour of exaggeration; but when I read of the horribly defective and disgusting condition of the so-called drainage of houses which existed in this country less than a century ago, I am not surprised that even so acute an observer and thinker as Budd was led to believe that infection could be conveyed from sewage through the air, or, indeed, that the large majority of practitioners were firmly convinced that fevers were generated from filth. There was an excellent excuse for their belief.

In the address on sheep-pox Budd discusses the way in which the infection was probably brought from London to Allington, either by infected sheep driven along unfrequented tracks on the downs, or by fleeces in barges on the Kennet and Avon canal. As regards the extension of the disease from Allington to the neighbourhood, he considers various agents, men, sheep, flies, starlings and the air. All this was quite new to the practitioners of those days.

The exigencies of time and space will not allow me to discuss in detail all Budd's conclusions and suggestions. He was of opinion that in scarlet fever the desquamating cuticle was infectious, and he anticipated Curgenven and Milne in the belief that by anointing the skin with oil impregnated with some antiseptic (he used camphor), the infectious agent could be destroyed, and the patient allowed out of isolation before the completion of peeling. He recognized that the convalescent typhoid patient might be a source of infection, that is to say, he recognized the existence of typhoid carriers. This was in 1873. The existence of carriers in

[1] Snow and Budd were not alone at that time in holding that the infection of cholera was contained in the stools. I find the fact stated in some excellent articles on *The Contagion of Asiatic Cholera*, by Mr. E. O. Spooner, of Blandford, in the *Prov. Med. and Surg. Journ.* for 1849, pp. 34, 62 and 91.

cholera had, however, been suggested by Daubeny, of Oxford, in 1855, and by Hallier, of Jena, in 1867. Budd was, I believe, the first to make use of spot-maps in order to follow more easily the course and distribution of an epidemic. He employed them in the cholera epidemic in Bristol in 1866. In discussing the question whether pig-typhoid and human-typhoid are the same disease he makes the point, by no means unimportant in his day, that "to establish identity in a case like this it is necessary not to show pathological resemblances merely, but that the germ of one species of fever is capable of producing the other." He pointed out the necessity of boiling milk and water used for drinking.

The course of time has shown that some of Budd's conclusions are erroneous. It is unnecessary to particularize them, as they are quite obvious to the reader of to-day. They were due partly to the limitations of the knowledge at his disposal and partly to lapses of judgment, to which the possessor of the divine gift of enthusiasm is so prone.

As it is my business this evening to deal with Budd and his writings solely from the epidemiological point of view, I will do no more than record my opinion that, judging from the evidence he gave before the Royal Sanitary Commission in December, 1869, and from statements made in a paper he read at the Bristol Congress of the National Association for the Promotion of Social Science in September of the same year, he was prominent in a small band of men who had very strong convictions as to the necessity of a properly organized public health service and a clear conception of the essentials of such a service. He would have made an admirable medical officer of health, or, indeed, chief medical officer to a Ministry of Health, unless an excess of enthusiasm is to be considered as too dangerous a qualification for the tenancy of those offices.

I may summarize Budd's contributions to epidemiology by saying :—

(1) That he showed the absolute necessity of admitting the fact that infection plays a part, and an important part, in the spread of a certain group of diseases, and so dealt a shrewd blow to the old and erroneous doctrines of miasms and spontaneous generation, thereby helping to pave the way for an acceptance of the germ theory of disease ;

(2) That he was the first to point out comprehensively, and at the same time succinctly, the methods of investigation which were most likely to lead to the solution of certain important epidemiological problems ;

(3) That he was the first in this country to demonstrate the infectiousness of typhoid fever and to insist upon the importance of recognizing that fact ;

(4) That he was the first to show that the infection of typhoid fever could be conveyed by water ; and

(5) That he pointed out the infectiousness of consumption at a time when possibly not a single person in this country believed in it.

During his life Budd came to be regarded as a leading authority on infectious diseases, epidemiology and questions of public health. Nearly all his measures for dealing with an outbreak of cholera were recommended for adoption by the Commissioners appointed to inquire into the cholera epidemic in Northern India in 1861. He was offered by the Government a seat on the Royal Commission which investigated the rinderpest epizoötic, but he was obliged to decline it on account of the pressure of his professional engagements. Although for years he urged his views in the face of much opposition—they were stigmatized as "immoral," "illiberal," and "inhuman," by some of those who opposed them—and in spite of the fact that he was held generally in the highest esteem, yet, as he himself has told us just before he was laid aside, he had not succeeded in converting to his views any large proportion of his professional brethren. He had, however, the satisfaction of knowing that he had converted Sir John Simon and the General Board of Health, as well as several eminent physicians, amongst them his former teacher, Sir Thomas

Watson. In the course of time his doctrines became generally acknowledged. It is difficult to understand, therefore, why his work has of recent years been so forgotten. I take it, however, it is partly because the principles he enunciated and so consistently preached have for so long been accepted as true and absorbed into the common stock of knowledge. To a certain extent, too, much of his work required to be and was clenched by the discoveries of the bacteriologists, and though *finis coronat opus*, it has been too frequently forgotten by whose aid much of the work thus crowned has been accomplished.

[For help in the preparation of this paper I am much indebted to several gentlemen, and especially to Mr. A. J. Wright, F.R.C.S., surgeon to the Throat and Nose Department of the Bristol General Hospital, a grandson of William Budd, who supplied me with a list of the latter's writings, to which I have been able to add but little, and with information on various points in connection with his grandfather's life. Sir Arthur Newsholme, K.C.B., was so good as to lend me, with other papers, a copy of the pamphlet, No. 25 in the list, which I had not been able to procure. To these gentlemen I tender my hearty thanks.]

List of Papers, etc., by William Budd, M.D. Edin., F.R.S., arranged in order of Publication.

I.—On Infectious Diseases, etc.

1.—Malignant Cholera: its Cause, Mode of Propagation, and its Prevention, London, September 27, 1849. 2.—Cholera: its Cause and Prevention, *Assoc. Med. Journ.* (*Brit. Med. Journ.*), 1854, ii, 928, 950, 974, 1152, and 1855, i, 207, 283. (Letters; the first four are signed "Common Sense," the last two "William Budd." In the fifth letter Budd acknowledges that he wrote the first four.) 3.—Mode of Propagation of Cholera, *Lancet*, 1856, i, 379, and *Assoc. Med. Journ.*, 1856, i, 259. 4.—On the Fever at the Clergy Orphan Asylum, *Lancet*, 1856, ii, 617. 5.—On Intestinal Fever: its Mode of Propagation, *Lancet*, 1856, ii, 694. 6.—The Cholera at West Ham, *Brit. Med. Journ.*, 1857, ii, 955. 7.—Intestinal Fever essentially Contagious, etc., *Lancet*, 1859, ii, 4, 28, 55, 80, 131, 207, 432, 458. 8.—On Intestinal Fever, *Lancet*, 1860, ii, 187, 239. 9.—On the Contagion of Yellow Fever, *Lancet*, 1861, i, 337. 10.—Typhoid Fever and the General Board of Health, *Lancet*, 1861, i, 630. 11.—Observations on Typhoid or Intestinal Fever; the Pythogenic Theory, *Brit. Med. Journ.*, 1861, i, 457, 485, 523, 549, 575, 604, 625. 12.—On the Occurrence (hitherto unnoticed) of Malignant Pustule in England, etc., *Lancet*, 1862, ii, 164 (Abstract of Papers, No. 13). 13.—Observations on the Occurrence of Malignant Pustule in England, *Brit. Med. Journ.*, 1863, i, 85, 110, 159, 237, 316. 14.—Additional Note on the Occurrence of Malignant Pustule in England, *Brit. Med. Journ.*, 1863, i, 557. 15.—Variola Ovina, Sheep's Small-pox; or the Laws of Contagious Diseases illustrated by an Experimental Type, *Brit. Med. Journ.*, 1863, ii, 141. 16.—Investigation of Epidemic and Epizoötic Diseases, *Brit. Med. Journ.*, 1864, ii, 354. 17.—Memorandum on the Propagation and Prevention of Asiatic Cholera, Appendix III to the Report of the Commissioners appointed to inquire into the Cholera Epidemic of 1861, Calcutta, 1864. 18.—Bacteridia and Malignant Pustule, *Lancet*, 1865, i, 47 (Letter). 19.—Observations on Typhoid (Intestinal) Fever in the Pig, *Brit. Med. Journ.*, 1865, ii, 81. 20.—Memoranda on Asiatic Cholera, its mode of Spreading and its Prevention, Bristol, 1865. (Pamphlet: contains, amongst other matters, a reprint of Budd's memorandum to the Special Commission to inquire into the Cholera Epidemic of 1861 in Northern India.) 21.—The Siberian Cattle Plague, or the Typhoid Fever of the Ox, *Brit. Med. Journ.*, 1865, ii, 169. 22.—Cholera and Disinfection: Asiatic Cholera in Bristol in 1866, *Brit. Med. Journ.*, 1867, i, 413. 23.—Memorandum on the Nature and Mode of Propagation of Phthisis, *Lancet*, 1867, ii, 451. 24.—Scarlet Fever and its Prevention, *Brit. Med. Journ.*, 1869, i, 23. 25.—Can the Government further beneficially interfere in the Prevention of Infectious Disease? London, 1870. (Pamphlet: Reprint of paper read at the Bristol Congress of the National Association for the Promotion of Social Science, September, 1869.) 26.—Evidence before the Royal Sanitary Commission (December, 1869). Second Report of the Commission, 1871, iii, Part I, p. 42. 27.—Typhoid Fever; its Nature, Mode of Spreading and Prevention, London, 1873. (Book, roy. oct.; contains portions of previous papers on typhoid with some additional matter.) A new edition of this work has recently been published (1931) by the Delta-Omega Society, of New York; it contains a portrait of Dr. Budd.

II.—On Non-infectious Diseases.

28.—Contribution to the Pathology of the Spinal Cord, *Trans. Roy. Med. Chir. Soc.*, 1839, xxii. 29.—Rheumatism, Tweedie's *Library of Medicine*, vol. v, 1841. 30.—Gout, id., *ibid.* 31.—On Diseases which affect corresponding Parts of the Body in a Symmetrical Manner, *Trans. Roy. Med. Chir. Soc.*, 1842, xxv. 32.—Remarks on the Pathology and Cause of Cancer, *Lancet*, 1841-42, ii, 266, 295. 33.—Retrospect of Anatomy and Physiology for the year 1843-44, *Trans. Prov. Med. and Surg. Soc.*, xiii, 1845 (new series, vol. i), 43. 34.—Researches on Gout, *Trans. Roy. Med. Chir. Soc.*, 1855, xxxviii.

35.—Cases of Bronzed Skin, *Brit. Med. Journ.*, 1856, i, 41. 36.—The Frog as a Detector of Tetanus Poison, *Lancet*, 1856, i, 90. 37.—The Employment of Calcareous Salts in the Treatment of Rickets and other Morbid Conditions, *Brit. Med. Journ.*, 1857, i, 514. 38.—Sugar and Diabetes: a Case, *Brit. Med. Journ.*, 1857, ii, 943. 39.—Sugar and Diabetes: further Report of the Case of Samuel Snailum, *Brit. Med. Journ.*, 1858, i, 208. 40.—Hydatid Cyst of Liver; Withdrawal of Fluid by Tapping: Permanent Collapse of the Cyst, *Brit. Med. Journ.*, 1859, i, 273.

I have omitted from the lists one or two short letters of no importance. I have been informed that Dr. Budd published a paper on the treatment of croup by warm vapour and emetics, but I have not been able to find it.

Budd reprinted some of his papers, in some instances in different forms and with slight alterations (Nos. 1, 13, 14, 15, 17, 21, 22, 24 and 25). The reprint of the paper on scarlet fever and its prevention (No. 24) went through at least five editions (fifth, 1871). I have not seen all these reprints.

On page 692 of the second volume of the second edition of his *Lectures on the Principles and Practice of Physic* (1845), Sir Thomas Watson refers to Dr. William Budd's views on a certain point connected with continued fever. I have not, however, been able to find any work of Budd's on infectious diseases which was published before 1849. Sir Thomas was personally acquainted with Budd, and was possibly referring to an oral communication. The reference may be taken as evidence that Budd was considered to be an authority on the continued fevers even at that time.

Discussion.—Dr. J. D. ROLLESTON, while congratulating Dr. Goodall on the excellence of his paper, which was the third important communication dealing with typhoid fever which he had made to the Section and its predecessor,[1] regretted that the term "forgotten" had been applied to Budd. It was true that in this country none of the textbooks of medicine, public health, or infectious diseases published in recent years mentioned Budd with the exception of Hamer and Hutt's "Manual of Hygiene" and Stallybrass's "Principles of Epidemiology." On the Continent, however, and in the United States Budd's work had received due recognition in textbooks of medicine, public health and medical history, as was shown in France by Achard's monograph on typhoid fever, and the articles by Widal and others in systems of medicine; in Germany by the textbook on infectious diseases by Jochmann and Hegler, Kolle and Hetsch's work on experimental bacteriology and infectious diseases, and Schottmüller's article on typhoid fever in Bergmann and Staehelin's system; in the United States by Cecil's textbook of medicine, Fitzgerald's and Rosenau's works on public health, and Gay's monograph on typhoid fever, as well as by the three principal modern works on the history of medicine, viz., the textbooks of Garrison and Castiglioni, and the second edition of Hirsch's *Lexikon*. The most striking proof, however, that Budd was not forgotten was the recent edition, limited to 800 copies, of his monograph on typhoid fever which had been published for the Delta Omega Society by the American Public Health Association as the first of a series of public health classics. While admitting that until a few weeks ago William Budd had been to him merely a name which he had seen in reading Murchison's work on the continued fevers of Great Britain, Dr. Rolleston considered it undesirable that British ignorance of so distinguished a physician should be emphasized. He also regretted that Dr. Goodall had made no mention of Bretonneau, whose priority in the recognition of the specificity and contagiousness of typhoid fever was acknowledged by Budd, although Bretonneau had not pointed out, as Budd had done, that the contagion was transmitted by the intestinal discharges.

Dr. GOODALL (in reply) said that his paper showed how the profession in the British Isles had failed to remember Budd as an *epidemiologist*; he must emphasize that point. References to Budd of recent years had been almost entirely confined to his views on the infectiousness of typhoid, with two or three scanty allusions to his paper on the communicability of phthisis. From Dr. Rolleston's references to foreign authors he (Dr. Goodall) gathered that they, too, were mostly concerned with the work on typhoid. But Budd's epidemiological work extended far beyond that disease, and it was that which had been forgotten. The speaker, therefore, did not feel inclined to alter the title of his paper.

[1] *Trans. Epid. Soc. London*, 1900, xix, 164; *Proceedings*, 1918, xii (Sect. Epid.), 15.

As regards Bretonneau, here again Dr. Goodall said he had not been concerned with foreign views on the infectiousness of typhoid. Budd, in his writings, fully recognized Bretonneau's work; but he had to convince his countrymen not by quotations from foreign authors but by the demonstration of facts which occurred within his own experience.

At this meeting a paper was also read by Lieut.-Colonel J. MORISON, I.M.S., on "Bacteriophage and the Cholera Problem."

Section of Medicine.

[*November* 24, 1931.]

DISCUSSION ON NERVOUS DYSPEPSIA.

Dr. Robert Hutchison: We must begin this discussion by defining our terms: (1) What is dyspepsia? (2) What is nervous dyspepsia?

(1) By dyspepsia I understand any discomfort during digestion which originates in the *stomach*. This definition is meant to exclude digestive discomforts which have their origin in other organs, such as the gall-bladder, appendix or colon, even although the stomach functions may be disordered, reflexly or otherwise, in many cases of disease in these organs. This means that a case of so-called "gall-bladder dyspepsia," for example, is not one of "dyspepsia" as above defined.

(2) The adjective "nervous" as applied to a dyspepsia has no very definite meaning. It certainly excludes, however, all dyspepsia due to organic disease of the stomach. On the other hand it is not synonymous with "functional" dyspepsia, for functional disorders of the stomach may be brought about by dietetic errors alone, although perhaps most cases of functional dyspepsia are of nervous origin.

I suggest that the term "nervous dyspepsia" should be used to cover three kinds of case:—

(i) Functional disorders of the stomach of *central origin*, in which the determining cause is some emotional disturbance or anxiety state. These cases we may speak of as *psychoneurotic*.

(ii) Functional disorders which result from fatigue, although I admit that the endocrines may play as large a part as the nervous system in the mechanism of production of such cases. This group we may speak of as *fatigue dyspepsia*, and although psychical disturbances often play a part in it, I suggest that fatigue as such may produce dyspepsia apart from mental perturbations.

(iii) A group in which, although no disorder of the gastric functions can be demonstrated, the patient complains of discomfort of one kind or another which he —or more often she—refers to the stomach. There are some writers, particularly, I think, in Germany, who would reserve the title "nervous dyspepsia" for this group, but I would prefer to speak of them as cases of "*psychogenic* dyspepsia."

I suggest these subdivisions of nervous dyspepsia for the sake of discussion, but in these nebulous matters it is well not to be too dogmatic.

The gastric functions, disorder of which may result in dyspepsia, are those of secretion and motility. These functions are largely controlled by a local nerve mechanism, but they are also under the dominion of the central nervous system, exercised through the sympathetic and parasympathetic systems.

It is well known, of course, that wide variations in the secretion and tone of the stomach are compatible with normal functioning, but when the normal mean is sufficiently departed from, dyspepsia results, or is at least made likely. Roughly

we may distinguish two groups of functional dyspepsia: (*a*) The hypersthenic, in which peristalsis, tone and secretion tend to be exaggerated, and (*b*) the asthenic in which they tend to be defective.

I would suggest that exaggerated nervous excitability tends to produce, through the vagus, hypersthenic dyspepsia, and that it is often met with in over-worked, highly-strung men; whilst depressing emotions and fatigue tend, through the sympathetic, to lower tone and secretion, and that this is the origin of many of the asthenic cases so often met with in women.

But may there also be a disorder of the sensory functions of the stomach? We must accept it as proved that the gastric mucous membrane is normally insensitive, but it is conceivable that if the threshold of consciousness is lowered, impulses proceeding from the stomach may be felt as dyspepsia. I would suggest that this is what happens in the psychogenic cases so often met with in visceroptotic women. It may be that some lowering of the threshold may also occur in the psychoneurotic cases, so that a diminution of gastric tone, for instance, which would not produce symptoms in a normal subject, is capable of doing so in a psychoneurotic. The relation of what I have called the psychogenic cases to the psychoneurotic cases, however, is worth discussing. For my own part I would not regard all the abdominal women in whom psychogenic dyspepsia is met with as psychoneurotics.

The symptoms of nervous dyspepsia are rarely extreme. Real pain is not met with except, perhaps, in some of the psychogenic cases. Usually the complaints are of discomfort, flatulencies, sinkings, fullness and so forth. Nausea is very common, but actual vomiting is rare. Wasting does not occur unless as the result of overdieting. It is also characteristic that the symptoms vary very much from day to day and are not much influenced by diet.

The diagnosis can only be arrived at by exclusion, after taking a detailed clinical history, and making a thorough physical and X-ray examination, and giving a test meal if necessary.

Treatment must be directed to removal of the cause if this is possible. In the psychoneurotic group this involves the help of psychotherapy; in the fatigue cases rest is the first essential—in bed in the severe type, or by a holiday in the milder.

Pending the removal of the cause, or, in those cases in which, though known, it cannot be removed (e.g., financial anxiety), symptomatic treatment is justified. In the hypersthenic type this will take the form of a bland diet, antacids, bromides and belladonna; in the asthenic cases of a diet of small, dry meals, with the use of tonics and physiotherapy.

Treatment of the psychogenic cases is a difficult problem which we may discuss with advantage. As there is here no demonstrable disorder of function, symptomatic treatment is out of place. What help, if any, psychotherapy can afford it is for our psychotherapeutic colleagues to say.

Dr. T. A. Ross: I would not regard all the visceroptotic women as psychoneurotic, though I would regard them as psychogenic. In most cases I think this correct. The majority of these patients whom I have seen, whom Dr. Hutchison has so ably described in his paper on the chronic abdomen, suffer, it seems to me, rather from a psychosis than from a neurosis. The fact that treatment directed to their symptom will often produce temporary improvement does not invalidate this statement. These women whom we are now discussing differ, I think, in one important point from those dyspeptics whom we may label psychoneurotics, in that if you try to explain their symptoms on the basis of a nervous ætiology you encounter the greatest scepticism which, if you persist, develops into anger and hostility. Dyspeptics whom I would describe as psychoneurotic, on the other hand, will at least listen to and be interested in a

description of how it is known that mental states affect gastric function. They can be shown that fear of food, or rather fear of what will happen if they eat certain things, produces the same results in their stomachs as those produced in Alexis St. Martin's stomach when he was depressed about anything. Approach those others with this same story and you can get a very different reception. Such a tale, they maintain, is wholly inapplicable to them. They have adhesions, they have loose sigmoids, they have one thing or another. And as has been said, persistence on the doctor's part in presentation of the functional view leads merely to anger. This is the very hall-mark of delusion—a phenomenon quite different from that of fear or doubt.

The delusion differs in this way from the phobia. You may not abolish a phobia by talking about it, but the patient will allow you to talk. The subject of delusion will not permit you to waste his time. As has been stated, there is nothing against this view in the fact that these patients are temporarily benefited by yet another operation. The satisfaction to be derived from severe punishment is frequently obtainable by psychotics as well as by psychoneurotics.

I have dwelt on these conditions at some length, because I do not think that psychotherapy is of much use in these psychotic states. I do not wish to label every woman with the chronic abdomen as a psychotic, but I have no doubt many of them are. The genuine anxiety-neurosis patient does not hanker after operations. These patients undoubtedly do. I fear, therefore, that I am unable to throw any light on the treatment of psychotic dyspepsias.

On the question of fatigue I do not expect to find myself in so much agreement with general physicians. We cannot burke the question, "What is fatigue?" and it seems that nobody is very sure. For most people it means that the quantity of energy discharged has been so much in excess of that taken in, that potential has been lowered, and therefore work can no longer be done efficiently. That that end-result has been achieved is probable enough, but my point is: "Has there been any loss of quantity of energy? Is it not rather that the patient feels exhausted because he is refusing to turn on his energy, of which he has abundance? Are we dealing with a real loss of storage of energy, or are we dealing with someone who has plenty of energy if he but knew how to use it?" The distinction is not academic, for it seems to me that we may put such a patient to rest for a very long time without achieving anything in the way of making him feel less exhausted. While he is in bed he may feel very well, and the dyspepsia will probably diminish. But unless he has received some instruction which will induce him to take a different view about exhaustion from that which he had before, his ultimate gain from rest in bed will not amount to much. Nay more, unless he has received instruction before he went to bed, the dyspepsia will not be much better while he is there. Most of these patients when advised to go to bed will say that unless they have some exercise, exhausted though they are, they will not be able to digest. They must then be told something like this, that exhausted as they are, they will digest better if their energy is not diverted from their digestion. If they accept this all will go well. But what has helped them? The rest in bed, or the psychotherapy?

My view is, then, that the exhaustion is not the cause of the dyspepsia. Both are the products of anxiety, and it is anxiety that we are called on to treat rather than either exhaustion or dyspepsia. This may be done in the way of palliation, or by the attempt at radical cure. The latter may involve a long analytical investigation, and for enormous numbers of these patients this is probably at present, at least, impracticable. A less ambitious psychotherapy is, however, of considerable value. The general mental state of the patient can be helped by sympathetic conversation over his general difficulties. All these patients feel that they are fighting a lonely battle, that no one cares to understand their difficulties, and the mere knowledge that someone does understand what a difficult time they

are having and will try to help them is of great assistance. These patients are the subjects of many fears, of which they do not talk unless encouraged, and among these are the fears of what the symptoms of which they complain may lead to. They fear that if they do much their exhaustion may become overwhelming. They fear that if they eat this, that, or the other, their dyspepsia will become worse. Every meal is eaten in fear of the result, therefore every meal is followed by indigestion. If time and care are taken in the explanation, most of them can be persuaded to eat anything without trouble. To my mind this is the explanation of the success of a planned diet and of most of the drugs which are of benefit. The patient is told by the doctor either implicitly or straight out that benefit will result. His anxiety about his digestion is thereby relieved. He has placed the reponsibility on someone else's shoulders, and therefore it is no longer on his own. In the history of the treatment of dyspepsia we have had such brilliant results from such different diets which have their day and disappear. We have different doctors getting such good results from such different régimes that no other explanation seems tenable.

If one wishes, because of lack of time, to confine one's inquiry into the symptom in question, one is frequently compelled to go much further along the road into a general analytical investigation. A woman, aged 30, complained of indigestion and exhaustion among a host of other symptoms. She made much of the indigestion, but was singularly unwilling to let me explain anything about it, not that she objected to my view, but having herself said that her dyspepsia made life not worth living, she turned round and said that time was short and there were much more important things to talk about. Why waste time over indigestion? It struck me that she was defending herself from talking about it, and I was persistent. It quickly took her back to childhood and girlhood, when every meal eaten at home had been distressing. The mother wished the family to "eat like ladies and gentlemen," the father would eat not only peas but other things with his knife; the mother openly jeered at his table manners. They were wholly unable to have friends in to meals because of father's bad manners. There were other reasons for meals being sources of anxiety, and not only this girl but most of the others suffered from dyspepsia, a subject of perpetual discussion in that household. Now here we were brought straight up to the whole complex relationships between children and parents, for of course there were many things to be talked about besides meals, which the patient had resolved when she came under my care not to talk about from motives of loyalty to her parents. Perforce we were driven to a much more radical treatment than had been intended. And the more we make our treatment radical, the more we shall realize that it is not extrinsic factors that are important in these matters. It is not diet, or fatigue, or financial difficulty, or adversity of any kind, which determines whether a man shall have neurotic symptoms. Why does one man stand up in the firing line for years and another becomes a nervous wreck in a few days? Why does one man face financial trouble without symptoms and another become dyspeptic? It is because one has learned to face difficulty with equanimity more than another. And it is the hope of psychotherapy that it will not so much concern itself with the relief of symptoms, but will enable people to acquire equanimity in greater degree.

Dr. H. Bedingfield: Cases of nervous dyspepsia superficially appear to be similar in nature, but I believe it useful, from a practical as much as from a theoretical point of view, to recognize that they may differ from each other in their nature, genesis and evolution.

We can differentiate a mechanical dyspepsia, in which symptoms arise from inability of a fatigued or congenitally feeble musculature to deal with food material beyond its capacity, and a dyspepsia of chronic intestinal invalidism or so-called

visceroptosis. What remains represents more accurately, to my mind, true nervous dyspepsia.

Its chief characteristics are:—

(1) There is no typical history of a childhood disturbed, beyond the average, by he physical disorders of early life.

(2) Digestive symptoms, apart from early infancy, do not as a rule make their appearance until after a puberty generally uneventful.

(3) There is no failure of energy, either mental or physical. Rather, indeed, relief is sought because the symptoms are interfering with an unimpaired capacity for work and play.

(4) There is no permanent disturbance of nutrition. I expressly say "permanent," because not infrequently it is a fairly rapid loss of weight which sends the patient to seek advice. Yet with successful treatment, weight is quickly regained. On the other hand, many cases, no matter how severe their symptoms, never show any marked deviation from their average standard.

(5) Instead of a diminished resistance to infection, there appears to be an actual increase, as evidenced by the infrequency and mildness of pyrexial attacks.

Time does not permit a detailed analysis of the clinically observed cases which lead me to this conclusion and I must here content myself with briefly summarizing their salient features.

In the first place the condition is apparently as common in men as in women. In fact, each year since the war the proportion of men to women has steadily increased, until now my total figures show a slight increase of men over women. The only apparent influence of sex is that in women the average age at onset is roughly 18 to 20, while in men it is 25 to 30, and that it appears to be more common in married men than in single, but in women more common among the unmarried. Social status, work, or profession, have no direct ætiological significance.

The digestive symptoms slightly differ from those met with in the chronic intestinal invalid, in that attacks of vomiting, nausea, and pyrosis are much more frequently complained of; that where pain is a marked feature, it tends more frequently to be localized somewhere in the upper abdomen than in the lower; that severe symptoms are often present with regular bowel movement, and that while mucus is frequently noticed in the constipated stool, typical attacks of muco-membranous colitis are quite rare.

When, however, these cases are further analysed it is found that nearly all fall into one of two groups. In one, where the symptoms are intermittent, alternating often with long periods of good health and unaccompanied by any pronounced signs of autonomic disturbance. In the other, symptoms once appearing tend to persist, fluctuating in intensity, and invariably accompanied by signs and symptoms of nervous and emotional instability. The associated symptoms of this second group are most frequently fibrositic pains, headaches and vertigo, chilly or burning sensations n different parts of the body, hot flushes, local blotching of the skin, and tremor. Palpitation, with or without tachycardia, is invariably complained of at some time or other, and premature contractions frequently cause great distress.

Physical examination does not reveal any departure from the normal to account for the symptoms. The secretory response of the stomach and the motor functions of the intestinal tract show nothing characteristic except phases of irritability, spasm, or hypotonia. In short, these cases present a functional disorder of the gastro-intestinal tract in its purest form and for that reason present a more puzzling ætiological problem than does even the chronic intestinal invalid. They were inexplicable as long as pathology remained cellular and mechanical.

The first light to be thrown upon the subject coincides with the passing of pathology from the cellular to the molecular, and from the "era of specific ætiology" to the idea of constitutional differences in reactivity associated primarily with the names of Eppinger and Hess.

Their original theory, lacking the confirmation of clinical experience, found modification in the idea of an inherited vegetative imbalance. This in its turn is no longer tenable in face of the extensive investigations undertaken by Petersen and Levinson on the "Skin Reaction, Blood Chemistry and Physical Status of Normal Men and Clinical Patients." Their elaborate and exhaustive work has led them to conclude there is little justification for trying to establish the existence of a purely endocrine imbalance, and less so of a purely nervous one. In other words, they emphasize the impossibility of trying to coördinate isolated observations on the vegetative and central nervous systems without a proper regard for all the other factors of the vegetative apparatus, namely, cell permeability, ionic equilibrium, etc.

This seems to me to be a remarkable confirmation of the fertile concept of Kraus who regards every individual as the resultant of the interaction of two distinct personalities; one, the phylogenetically long established, the so-called "Tiefenperson," i.e., the vital individuality of the primitive protoplasm, represented by the molecular side of the vegetative apparatus; the other, the centrally organized and much younger cortical personality, the mind, with the vegetative and central nervous systems as the pathways of communication between the two.

Health may be defined simply as adjustment between the individual and his environment, and this in turn depends upon an integrated deep and cortical personality. This integration may be upset by disturbance in the deep by inherited, congenital or acquired defects. So far no one has been able to demonstrate such as the basis of neuroses and, until they are demonstrated, it is legitimate to look to the cortical personality for evidence of disturbance.

The latter functions at three levels of evolutionary development, vegetative, sensorimotor and psychical. In infancy the disorders of cortical personality or, for short, functional disorder, manifest themselves almost entirely through the vegetative apparatus, particularly in that part controlling assimilation and excretion. A moment's reflection will show why this part should be the most frequently disturbed. Nutritional satisfaction is a function not only of the tiefenperson but of protoplasm in general, appearing at the very moment the lowest protozoa are organized to take in nourishment with their whole undifferentiated unicellular bodies. In other words, the urge to feed and the satisfaction of satiation are antecedent to the formation of a cortical personality, and remain co-existing with the latter as we know from the physiological changes which occur in body cells and fluids after a satisfying meal. That is to say, at the dawn of mental life there exists a mature ready-made psycho-physiological mechanism to reflect disturbance in the development of integration.

This may be brought about in one of three ways: (1) By unsuitable alimentation; (2) By some congenital or inherited defect in the primordial protoplasm rendering appropriate food difficult of assimilation; (3) By lack of harmony in the development of cortical personality.

As the first two do not appear to play any definite ætiological part in pure nervous dyspepsia they will not be considered further, and we pass on to consider the third situation.

The harmonious development of the cortical control depends largely on the emotional influences which play around the child from its first hour until it passes out of parental control. Any disharmony will be reflected physiologically in the gastro-intestinal tract as being the only psycho-physiological mechanism available.

What are the emotional disturbances which tend to make their appearances at such an early age? In a final analysis they must depend upon the constant conflict going on between the instinctual basis of the mind and the ego, or in other words, between the deep personality and the cortical. The ego, which is the nucleus of mind as we mean it in contrast to matter, appears to be largely an

implantation from without, a seed sown by the environment, but nurtured and moulded by parental control. This is being constantly bombarded by instinctual impulses. Satisfaction of these impulses would be pleasurable to the organism, but not so to the ego, in that they conflict with the pattern which is being imposed upon the child. Hence at each stage of development, pregenital, genital and post-genital, adjustment must be found which compromises between the ego and the instinctual impulses. In the pregenital stage, if this compromise is not satisfactory, the maladjustment, as we have shown, reflects itself in the gastro-intestinal tract. The more frequently this occurs the more readily will later maladjustments tend to use the same path as the result of conditioning.

This compromise can be brought about in one of three ways. Each way is equally satisfactory in dealing with the immediate situation, but not so regarding any future one.

In the first place the conflict may be resolved along the lines of sublimation; that is to say, the instinctual impulses may be so radically and intrinsically changed before impinging on the ego that they cease for all time to worry it. In such a case there is a total absence of functional disturbance.

Secondly, instead of being sublimated, they may be more or less completely absorbed, but only after the ego has reacted to them with a feeling of guilt, fear, or anxiety, based on the type of environmental reaction. Hence in this case they represent not only the positive unconscious impulses, but also the elements of ego-reaction against these impulses. This appears to be the genesis of the neurotic character or nervous individual, as contrasted with the individual with a neurosis. Finally, failing one or other of these mechanisms, the process of repression may be utilized, whereby the instinctual impulses, neither sublimated nor absorbed, are kept at bay out of harm's way so long as repression is effective, but ready to reassert themselves—distorted it is true—whenever repression breaks down.

As we have already seen, the first satisfaction enjoyed by the ego must be alimentary in nature, but it is only apprehended by the ego in terms of the mechanisms which begin and end the process, namely, incorporation by the mouth and rejection by the anus. Apart from this, the process of digestion and assimilation remains completely outside consciousness.

Now any discord resulting from the above-mentioned conflicts will appear first as a simple disturbance of these mechanisms, in the form of vomiting, constipation, etc. Continued or severer disturbance will lower the threshold, until finally the functions of other parts of the gastro-intestinal tract rise into consciousness to be perceived as pain, discomfort, etc. Although these physical consequences of malfunction may require symptomatic treatment, the ego, intolerant of conflict, is itself resolving the cause of them in one of the ways mentioned above, either by absorption or repression.

In the first, as we have seen, the reactions of fear and anxiety are absorbed into the personality along with the instinctual impulses. This tends to mould the individual into one with either a generally timid nature or an extremely cautious one. Both types deliberately avoid situations in life in which the emotional content recapitulates the early conflicts. Such situations arouse the anxiety or fear associated with the primitive ego reaction and they seek refuge in flight. But few individuals, even the most leisured or the most cautious, can pass through life without at some time or other having to face such situations, and the reaction is at once on the primitive level with re-activation of their early gastro-intestinal conditioning. These are the individuals, otherwise healthy mentally and physically, who complain of simple anorexia, loss of weight, attacks of vomiting and bouts of obstinate constipation.

If the emotional situation is one easily and quickly dealt with, the symptoms pass away, but if not, the continuance of the symptoms brings about physiologically

and by later acquired mental associations, reflex disturbances in other parts of the gastro-intestinal canal, terminating in so-called gastritis, colitis, and possibly much clinically diagnosed chronic cholecystitis. Fortunately, these cases are easy to treat.

Once the patients are satisfied that there is no organic disease, they readily co-operate in eliciting their emotional difficulties. Having no strong repression to deal with, they are able, as a rule, to gain considerable insight into the mechanism whereby their symptoms are produced. Or, if they are not intelligent enough to co-operate but have sufficient time and money to isolate themselves from the difficulties in one or other of our health centres, the result will probably be the same. No doubt in earlier ages many cases of nervous dyspepsia found a complete cure in monastic life.

But the problem is a much more difficult one in the case of the individual whose symptoms depend on a failure of repression. The kind and quantity of the repressed material and the strength of repression vary so much from individual to individual that the life situations which may bring about release with symptom-formation are legion. When the repressed material is small and repression strong, prolonged emotional strain of an easily identified nature may be the cause, and the symptoms may be fairly easily removed, either by strengthening the repression or by a comparatively simple psychological analysis. On the other hand, when the repressed material is great, and includes the later repressions of the genital and post-genital stages of development, the individual may be quite unable to make any of the ordinary matter-of-fact social adjustments of daily life without symptom formation. Moreover, the primary patterns of rejection and retention themselves tend to be repressed, and only the secondary ones, in the form of localized pain or other symptoms, appear. In addition, in every case they are accompanied by signs and symptoms of a widespread vegetative disturbance involving, in some degree or other, functions outside the gastro-intestinal tract.

Here, too, institutional treatment may bring about a certain amount of improvement, but severe cases tend to relapse once they are exposed again to the strains of daily life. Analysis appears to be the only permanent cure, but where general medicine can help is in the relief of the consequences of the malfunction. For just as in the previous group, only much more so, continued malfunction brings in its train physical signs of its presence, in the shape of irritability, spasm, or atonia, often accompanied by secretory disturbance. Finally, there may result actual organic change, as the work of Rohdenburgh and others tends to show. In their investigations on so-called chronic appendicitis and cholecystitis they have clearly distinguished a pathological change, distinct histologically from inflammatory reaction, in which the first deviation from the normal is a surrounding of some of the cells of Meissner's ganglion by a small collar of round cells. These increase in number until the ganglion is completely buried. This cellular infiltration then recedes, to be replaced by a general fibrosis, accompanied by a varying degree of sclerosis in the blood-vessels. This productive change these workers consider to be the direct consequence of abnormal impulses passing through the vegetative nerves.

My conclusion is that all functional disorders of the gastro-intestinal tract fall into one of three groups, namely those found in : (1) Constitutionally inferior individuals. (2) Individuals of a neurotic character who respond to life's difficulties by way of the digestive system. (3) Those whose symptoms are the result of failure of repression.

Dr. J. A. Ryle said that the classification of such disorders as nervous dyspepsia was difficult because of their variety and their dependence upon a multiplicity of ætiological factors. Constitution, both mental and physical, environment and habit

might, severally or collectively, contribute to the production of symptoms. Nevertheless, the classification suggested by Dr. Robert Hutchison was valuable for clarifying thought. Might not another group of dyspepsias, due to nervous tricks or habits, be added? The habit of aerophagy was a very common cause of dyspepsia: it might be the sole cause or merely a subsidiary anxiety symptom. The patient's too frequent insistence on the necessity for "a diet" to cure his indigestion should be deprecated. The profession was too ready to attach importance to dietary measures. Quite comparable with the more urgent and severe condition known as "anorexia nervosa" was a similar, more chronic state of "dyspepsia nervosa," due largely to continued curtailment of necessary foods, loss of weight and energy, and anxiety of mind as to which foodstuffs were permissible or injurious.

Dr. Langdon Brown said that in general he agreed with Dr. Hutchison's division of nervous dyspepsias into hypersthenic and asthenic, and, while not accepting all Eppinger and Hess's views, he regarded the former as the result of an over-acting vagus, while the sympathetic produced the symptoms of the latter. The sympathetic did so, however, by a process of active inhibition of peristalsis and closure of sphincters. "Atonic dilatation" of the stomach need not be due to a loss of tone; it was far more frequently due to sympathetic inhibition from an emotional cause. He agreed with Dr. Ryle both as to the importance of air swallowing as a cause of nervous dyspepsia and as to the exaggerated importance which had been attributed to diet.

He referred to cases illustrating each of these points. He had been interested in Dr. Ross's description of the "chronic abdominal" patient who was a psychotic; previous speakers had referred to this type as if it only occurred in women; the worst case that he himself had seen, however, was in a man.

Dr. F. Parkes Weber said he would only allude to one aspect of the nature of the cases in question. In his opinion almost every case ordinarily labelled "nervous dyspepsia" should be regarded as the result of a disorder or exaggeration of the normal nervous protective reactions[1]; that was to say, of a vital protective mechanism which was of extreme importance for the maintenance of health and the attainment of longevity. To explain what he meant by this class of normal nervous reactions he instanced the case of a physician who, after a strenuous morning's work, unwisely tried to take an ordinary midday meal with his family—but in half the time taken by other members of the family—before leaving to keep an important early afternoon engagement. He vomited whilst in his carriage on the way. In his temporary state of mental strain he clearly ought not to have tried to take an ordinary meal in such a hurry. Those who under similar circumstances could eat a full meal without any untoward results were the bodily and mentally robust individuals who, if they habitually did that kind of thing, paid the penalty by unnecessary metabolic or degenerative disease and by death at about sixty years of age, whereas those who were "hampered" (as they often wrongly termed it) by troublesome nervous reactions—which were really defensive in nature—were more likely to remain healthy and live to ninety years of age.

Dr. E. Stolkind said that cases of neurasthenia and hysteria—"minor psychosis"—belonged to psychoneurotic groups. A great number of his own patients suffered from anxiety neurosis. On the other hand patients suffering from intoxication—morphine, cocaine, nicotine—should not be included in these groups.

He proceeded to quote details of some of his cases, illustrating the influence of anxiety in producing dyspepsia—anxiety as to pregnancy, anxiety as to organic disease and anxiety increasing the severity of the symptoms of organic disease actually present.

[1] For instance, the abnormal sensation of discomfort or pain, due, as Dr. Hutchison suggested, to a "lowering of the threshold of consciousness."

Dr. Louis Mirvish : It is not always easy to distinguish between nervous and organic dyspepsia. Many organic abdominal diseases have a large functional element superadded. Symptoms which for a long time were considered to be functional have later proved to have an organic basis, e.g., Reichmann's syndrome. The large number of partial gastrectomies performed in Germany in recent years has furnished fresh material for histological examination, and these studies have given rise to a body of opinion which regards all so-called nervous dyspepsia as forms of gastritis or other organic disease.

The symptoms produced by gastritis will vary according to its site, and with the type of individual. In the hypersthenic type pyloric gastritis or duodenitis will produce symptoms simulating duodenal ulcer. These patients very often show a circumscribed area of tenderness in the epigastrium—gastralgia—which is really a referred pain from the stomach. That such people are improved by a holiday, by removing sources of worry, or by psychotherapy, without restricting their diet in any way does not rule out organic disease, as similar results can often be obtained in nervous patients suffering from duodenal ulcer, colitis, or other organic disease.

Dr. Hutchison (in reply) said that the discussion had revealed more unanimity than might have been expected. He was particularly interested to note that the psychoneurologists were inclined to regard the cases which he had classed as psychogenic as really psychotic. Everyone would agree with Dr. Ryle as to aerophagy being of nervous origin, but he (Dr. Hutchison) had specially excluded it from the discussion as it was a symptom originating not in the stomach but in the œsophagus. For the same reason he excluded the cases in which the fats disagreed. The fault in these cases seemed to be in the liver rather than the stomach. He would regard the interesting cases described by Dr. Stolkind as hysterical, but it was doubtful whether hysterical vomiting could be included in the group of nervous dyspepsias. With regard to the large part attributed to gastritis in the production of dyspepsia in some German clinics, these cases might simulate organic disease such as ulcer or carcinoma, but it was unlikely that they would be mistaken for nervous cases, nor could he agree that the term nervous dyspepsia should be limited only to the psychogenic cases, as was the teaching of some German authorities.

The psychoneurological speakers were naturally inclined to attribute a large part to mental factors in the production of the " fatigue " cases. It was no doubt true that these factors might be present without the patient really being aware of them, but none the less cases did seem to occur in which physical exhaustion alone was capable of producing dyspepsia. As to the question of diet which several speakers had raised, he agreed with Dr. Ryle as to the inadvisability of over-dieting such patients, but in spite of this he believed that in those cases in which there was definite impairment of function—but not in the psychogenic group—reasonable dietetic restrictions were justifiable.

Section of Obstetrics and Gynæcology.

President—Mr. VICTOR BONNEY, M.S.

[*November* 20, 1931.]

Recent Advances in Deep X-ray Therapy in Non-malignant and Malignant Uterine Disease.

By L. MARTINDALE, C.B.E., M.D., B.S.

TECHNIQUE in deep X-ray therapy has developed rapidly during the last few years, due partly to improvement in the various types of apparatus and hot cathode X-ray tubes and partly to the extensive and world-wide clinical research into the end results of treated cases.

In this short communication the advances in the X-ray treatment of diseases of the uterus, both malignant and non-malignant, will be dealt with and illustrated by results culled from the literature on the subject and from the author's own experiences.

(I) *Non-malignant Disease of the Uterus.*

X-ray technique in the case of non-malignant diseases of the uterus, notably—

Glandular hyperplasia of the endometrium and fibromyomata of the uterus and adenomyomata, has undergone three stages in developments :

(1) That in use from 1902 to 1913, beginning, therefore, six years after Röntgen discovered X-rays for W. J. Morton, of New York, treated his first case of fibromyoma in 1902.

In 1904 Foveau, in France, published a cured case.

In 1913 Albert Schönberg submitted a series of 796 myoma and 354 metropathic cases to the Congress of Physiotherapy in Berlin, Krönig and Gauss's cases being the most numerous in that series.

(2) The technique used from 1913-1920 was known as "Crossfire small field method;" we worked at a lower voltage, used eighteen or more portals of entry, filtered through 3 mm. of aluminium and gave the treatment every three weeks—for four to six treatments in all—measuring the dose by means of Kienbock strips.

Béclère, of Paris, used larger fields, gave the treatment every week or fortnight, and measured the dose with Sabouraud pastilles.

(3) The next development, in 1920, came with the higher voltage in the 1° current; 0·5 mm. zinc was used as a filter, together with 2 mm. aluminium and only 4 fields or portals of entry were used. The results were as good and the treatment was given in one sitting, which lasted three and a half to four and a half hours. With further developments in the X-ray plant, notably the use of transformers in place of the old double induction coil and an improved X-ray tube capable of transmitting

heavier currents, the time of treatment was considerably shortened, and in 1927-29 a very perfect X-ray apparatus (the Stabilivolt) so reduced the necessary duration of the treatment that it has been possible to give a castration dose sufficient to ensure a permanent amenorrhœa, in a case of climacteric hæmorrhage, in from thirty-five to ninety minutes.

As an illustration of the extent to which this treatment has become common, a comparative table of cases of fibromyomata and metropathia hæmorrhagica, treated by deep X-ray therapy, up to the year 1927, was recently collected by Gauss and his collaborators.

TABLE I.

	Cases	Lost sight of	Cured	Relapse	Improved	Mortality	Failures	Amenorrhœa	Clinical cures
			In % after deducting cases lost sight of						
John:									
1913									
Ro. My	846	38	85·1	0·5	9·7	0·4	4·3	85·6	95·3
Ro. Me	338	18	80·6	1·2	14·1	—	4·1	81·8	95·9
Ro. My and Me	211	15	76·0	1·0	18·3	—	4·6	77·0	95·3
Schmid:									
1914-19									
Ro. My	2,728	114	84·0	1·8	9·2	0·15	5·5	85·8	95
Ro. Me	1,497	30	90·0	0·8	7·97	—	0·7	91·3	99·27
Ro. My and Me	994	22	93·3	0·1	2·98	—	3·6	93·4	96·38
Lamm:									
1920-21									
Ro. My	1,374	95	80·6	5·2	12·2	0·08	1·95	85·8	98
Ro. Me	659	22	94·9	0·9	1·9	—	1·26	96·8	98·7
Ro. My and Me	41	3	97·4	—	—	—	2·6	97·4	97·4
V. Ammon:									
1922-27									
Ro. My	6,967	527	91·4	1·3	2·7	0·1	4·5	92·7	95·4
Ro. Me	2,243	352	96·2	1·3	1·6	0·05	0·7	97·6	99·2
Ro. My and Me	1,452	99	98·2	1·4	—	—	0·3	99·6	99·6
	19,350	1,335							

Third column, cured = permanent amenorrhœa.
Fifth column, improved = those cases in which normal menstruation had taken the place of profuse menstruation.
Eighth column, amenorrhœa = includes the permanent amenorrhœa and relapses, and therefore denotes *primary cures*.
Ninth column, clinical cures = includes permanent amenorrhœas, relapses, and improved cases.
Ro. = X-ray therapy. My = myoma. Me = metropathia hæmorrhagica.

On examining these results we see: (1) The steady improvement resulting from the newer technique (*vide* "clinically cured" column), viz., from 95 to 99%; (2) the small percentage of relapses, only about 1% now occurring; and (3) the small percentage of failures, viz., 0·7 to 0·3% in the case of metropathic hæmorrhages, and only 4% in the cases of fibromyomata.

Deducting the cases lost sight of, there remains a total of 18,000 cases treated by deep X-ray therapy, in which the subsequent history has been followed up and recorded, so that we now feel justified in saying that this treatment has proved of value in 98% of cases, and without a primary mortality.

Contra-indications to this treatment are:—

(1) Uncertainty in diagnosis; (2) possible pregnancy suspected; (3) submucous fibromyomata; and (4) pedunculated subperitoneal fibromyomata; (5) urgent pressure symptoms; (6) if dangerous hæmorrhage is present; (7) degeneration of fibroid suspected, e.g., necrobiosis; (8) fibroid complicated by possible presence of sarcomatous or carcinomatous degeneration; (9) very large fibroids; and lastly (10) suspected complication of serious disease in other pelvic organs.

Consequently the number of cases safely X-rayed is a definitely limited one.

In examining the percentage of cases operated upon or treated with X-ray therapy, we find the following figures:—

TABLE II.

	Operated cases	Treated by X-rays
Franz: 1910-22	65%	35%
Seitz and Wintz: 1920	16%	84%
Zweifel—Doederlein: 1920	21%	79%
Gal: 1922	45%	55%
Lockyer: 1924	77%	23%
Martindale: 1925	56%	44%
P. Werner: 1927	50%	50%

To-day the author's percentage of treated cases is slightly different as seen in the following table:—

TABLE III.—AUTHOR'S CASES OF FIBROMYOMATA OF UTERUS AND METROPATHIA HÆMORRHAGICA, 1914 to October, 1931.

Treatment	No. of cases	Cured	Died
Surgical operation	260	98·1%	1·9%
Subtotal	156		
Total	53		
Abdominal myomectomy	35		
Vaginal myomectomy	16		
Intensive X-ray therapy	224	97% *	0
Radium	16	0	0
Total	500		

* Estimated on 218 cases up to March, 1931.

Although many of us have now adopted the "one treatment" method, Béclère and nearly all the French authors—B. Vignes, Béclère (junior), Solomon, de Brancas, and others—still favour giving the dose in a series of radiations, lasting over several weeks; the Italian gynæcologists, Spinelli and Bacialli, also adopt this method, for they believe that in this way a total smaller X-ray dose may be given.

Gauss, Wintz, Werner and others, however, also try to use the minimum necessary dose, but achieve this end in another way; they alter the dose according to the size of the fibroid, measurements of the pelvis, and so on. An attempt has also been made to regulate the hæmorrhage in profuse menstruation, without causing an amenorrhœa, and to do this Pope and Mansfeld first suggested and carried out radiation of one ovary only. Pope believes that in this way hyper- or dys-function of the ovary is corrected.

Table IV shows their results in this method of one-sided X-ray castration; we find that for fibroids and climacteric hæmorrhages the treatment is not certain enough to be advocated.

TABLE IV.

Preceded by diagnostic curetting			Without diagnostic curetting		
No. of cases	With result	Followed by amenorrhœa	No. of cases	with result	Followed by amenorrhœa
44	20	8	44	25	7
	without effect 16			without effect 12	

Temporary Amenorrhœa or Sterilization is attained with 28% of a unit skin dose. After treatment, menstruation takes place once or twice, in some cases even three times, but then ceases for from two or three years; after this it begins again, although usually the loss is then regular and normal in quantity. (The possibility of this X-ray amenorrhœa depends on the fact that the primordial follicles have less radio-sensibility than the older—the ripe and ripening Graafian follicles being 25% more sensitive than the primordial. The corpus luteum is less radio-sensitive than the Graafian follicles.)

This treatment was used by Fraenkel, Eymer and Menge, as long ago as 1912, for salpingitis and pelvic peritonitis, and in tuberculosis in which temporary amenorrhœa improved the condition of the patient and prevented the relapses prone to occur during menstruation. Several authors, notably Wintz, use it in carcinoma of breast after treatment of the original disease.

The author has proved its usefulness in rare cases of menorrhagia in younger women, in which no other treatment had resulted in a cure. In three cases the temporary amenorrhœa was followed some years later by healthy pregnancy and childbirth, the children being apparently normal and healthy.

Deep X-ray therapy, however, remains the treatment de luxe for climacteric hæmorrhage and small interstitial fibroids in women over 40, according to the opinion of the gynæcologists of the clinics mentioned, and others who have had experience of the excellent results following this treatment in carefully selected cases.

(II) *Malignant Disease of the Uterus.*

In a recent statistical report from the world literature on the treatment by radiation of carcinoma of the cervix, which has just been published in Veit-Stoeckel's "Handbuch der Gynäkologie," the results obtained by fifty-four authors in clinics in twelve different countries are recorded, the countries cited being America, Austria, Belgium, France, Germany, Hungary, Holland, Italy, Switzerland, Japan, Russia, and Sweden (the clinics mentioned in the first five countries having been visited in several cases more than once by the author of this paper). (See Table V.)

The technique in the various clinics varies. Taking examples of a few of those getting the best results in deep X-ray therapy, we find the following main differences:—

(1) *The new Freiburg method*: (*a*) After histological confirmation radium is given in the following dosage: 2,000 mg.h. intra-uterine, 1,000 mg.h. vaginally. (*b*) The parametria are X-rayed during the following weeks with 100% H.E.D. (viz., unit skin dose) (Neo-intensive Siemen's Reiniger Veifa apparatus, or Stabilivolt. 180 k.v., 30 F.D. 6 by 8 cm. field, 8 to 10 in all, and 0·5 Cu filtering). (*c*) Three weeks after the first radium treatment a second dose of radium, viz., 2,000 mg.h. intra-uterine and 1,000 vaginally, is given (at most 90 mg.h. for 33½ hours).

In addition, in suitable cases, a radio-active bi-diasporal 360 (Dr. Klopfer) is used.

For inoperable cases, three months after the first treatment, deep X-ray therapy, 100% H.E.D., is repeated. Should the temperature rise after the radium is introduced, it is removed and is not re-inserted until the temperature has fallen.

For carcinoma of the fundus: Operable cases are operated on by the abdominal or vaginal route; 100% H.E.D. X-ray therapy is then applied.

Inoperable cases are treated as carcinoma of the cervix.

After-treatment: Injection of arsenic in the form of solarson or optarson.

(2) *The Erlangen technique*: Seitz and Wintz used only X-rays at first, but later combined them with radium, the X-rays being, according to Wintz, the more important, and the radium supplementary.

Technique.—First coppering.—(*a*) A preliminary weak galvanic current is passed through the tumour, by means of a copper anode; an attempt is made to disinfect in this way the deeper structures.

TABLE V.

Author	Clinic	Publication and time	Year of treatment	Duration of follow-up (years)	No. of cases	Absolute cures %	Relative cures %	Operability	Mortality %
America									
Clark and Block	Philadelphia	*Amer. Journ. Obst.*, 1921	—	5	144	10·4	27·3	15·3	
Schmitz	Chicago	*Journ. Amer. Med. Assoc.*, 1925	—	5	103	14·5	50	17·5	
Ward	America	Ref. of Assembled Gyn: stat., 1926	1920	5	181	23·2	—	—	
Healey	New York Memorial	*Canad. Prac.*, 1924	1918-21	5	155	9	23·5	21·9	
		Amer. Journ. Roentgen., 1925		3-7	375	16·8	—	—	
Austria									
Adler—Schauta	Vienna	Strahlentherapie, 1931	1912-15	5-8	58	24	—	—	
France									
Regaud	Paris	1919 Report	—	5-6	85	10·6	—	—	
		1925 Report	—	4-5	180	14·4	—	—	
		1926 Report	—	5	201	12·4	18·5	40·3	
Germany									
Burger—Kronig	Freiburg	Strahlentherapie, 1922	1913-16	5	76	7·9	12 Gr. I	33	
							10·9 Gr. II	72·3	
Bumm	Berlin	*Münch. M. W.*, 1926	1913-19	5	805	15·27	28·3	22·3	
Doederlein—Voltz	Munich	*Arch. of Gyn.*, 1927	1913-23	5-15	1,448	15·3	29·4	37·7	
Eymer—Menge	Heidelberg	*Zb. f. Gyn.*, 1925	1913-19	5	203				
Kehrer	Dresden	*Arch. Gyn.*, 1922	1915-17	5	129	27·9	40·7		
Lahm	Dresden and Chemnitz	Strahlentherapie, 1928	1915-23	5	469	23·5	40·7 (I)	12·5	
							34 (I and II)	43·2	
Maier—Pankow	Freiburg	Not yet published	1913-18	Kronig 5	112	10·7	27	33 Gr. I	
			1919-24 = 1913-24	Opitz 5	165	29	46·6	45 Gr. I and II	
Seitz	Frankfurt	*Klin. Wschr.*, 1922	1913-17	5	58	20·7	50	13·8	
Schmidt—Gauss	Würzburg	*Zts. Geb.*, 1923	1913-16	5	126	23·2	19·2 (Ra only)		
							5·9 R + Ro	48·4	
v. Seufert	Munich	*Msch. Geb.*, 1920	1913-14	5	205	19·5	39·2	50·0	
Scholten—Voltz	Munich	*Acta Rad.*, 1926	1912-20	5 (over)	1,260	14			
Warnekros	Berlin	*Merh. Ges. Gyn.*, 1920	1912-16	4-5	173	22·5	33	55·5	
Wintz	Erlangen	*Ebenda*	1913-23	5-15	740	18·9	57·4	14·6	
Wille—Franz	Berlin	*Zb. Gyn.*, 1922	1915-20	5	190	12·1	39·5	28·5	3·7, 2·6
Winter	Munich	Strahlentherapie, 1927	1918	5	48	8·3			
Zweifel	Munich	,, 1927	1926-27	5	3,969	16·9			
			1927	3	1,839	11·4		1·19	
Holland									
den Hoed	Amsterdam	,, 1928	1915-20	5	109	11	52·2	14%	
Hungary									
Lebroczky—Semmelweiss	Budapest	*Zb. Rad.*, 1926	1913-19	5	286	14·7	Gr. I 38	21·8	
							Gr. II 12·5		
Gal	Budapest	Strahlentherapie, 1928	1919-21	5	133	10·5	33·3	11·2	
Italy									
Bolarfio									
Japan									
Ikeda	Saga, Japan	*Zb. Gyn.*, 1927	1915-20	5 (more)	254	45·6			0·4
Russia									
Polubinsky	Petersburg	*Zb. Gyn.*, 1925	1914-21	5 and more	302	20·0	32		
Sweden									
Heyman—Forsell	Stockholm	Strahlentherapie, 1925	1914-18		217	19·8	40-50	66·6	
		,, 1926	1914-19	5	234	16·7	44·4		
		,, 1928	1914-21		541	21·6			
Switzerland									
Beuttner	Zurich	*Schweiz. Med. W.*, 1923	1914-18	5-8	189	17·5	44·4	12	
				7-10		12·7	47·2	27	

(*b*) Radiation of the primary tumour with 6-7 fields ; 6 by 8 cm. at 23 cm. F.D. and 0·5 zinc + 1 aluminium filtering. This dose = 110 to 120% H.E.D. radium 1,000 to 2,000 mg.h. If more than 1,000 mg.h. of radium is used, Wintz uses only 70-80% H.E.D. X-ray therapy.

(*c*) Radiation of parametria : 6-8 weeks after : 4-6 abdominal fields, 6 by 8 cm., 23 F.D. 0·5 Cu : 4-6 sacral fields and two lateral.

(*d*) At the end of one year, prophylactic radiation 70-80% H.E.D., in two large fields, is given.

TABLE VI.

Recent statistics have been published by the Erlangen Clinic to show the advances due to improvement in technique.

IMPROVEMENT IN METHOD.

Carcinoma of Cervix.

(After five years).

Symptom-free and able to work according to age.

Operable Cases.

Group I.	1916-1921 (77 patients) ...	56%
Group II.	1922-1926 (57 ,,) ...	68%

Inoperable Cases.

Group I.	1915-1921 (472 patients) ...	11%
Group II.	1922-1926 (315 ,,) ...	15%

IMPROVEMENT IN METHOD.

Carcinoma of Corpus Uteri.

(After five years).

Symptom-free and able to work according to age.

Operable Cases.

Group I.	1916-1921 (35 patients) ...	63%
Group II.	1922-1926 (45 ,,) ...	75%

Inoperable Cases.

Group I.	1915-1921 (43 patients) ...	2%
Group II.	1922-1926 (22 ,,) ...	23%

(3) *The Doederlein-Voltz method* (Munich).

First treatment.

Day
1. Preparation of patient, history taking, etc.
2. Pituitary radiation.
3. X-ray treatment.
4. Rest.
5. Diagnostic operative measures, excision, abrasion, fulguration, cautery, or ionization.

Day
6. Rest.
7. Treatment with radio-active substance (radium or mesothorium).
8. Rest.
9. Discharge of patient.

Second treatment eight weeks after.

Day
1. Admission, examination and preparation of patient.
2. X-ray treatment.
3. Rest.

Day
4. Treatment with radio-active substance.
5. Rest.
6. Discharge of patient.

And again eight weeks after if necessary.

X-ray treatment.

Up to 1922, many fields method. 6 fields 6 by 8 cm., 3 abdominal, 3 dorsal, at 23 cm. F.D. Also 1 vulval field at 30 cm. Filtering 0·5 zinc + 3 aluminium 180 k.v. 2·5 ma. in the 2° current.

After 1922, large field method. 10 by 15 cm. at 50 to 60 cm., 200 k.v. at 2·5 ma. on each field 90% H.E.D.

If possible this dose is given on one day, if not on two consecutive days. If the Baudelocque measurement is more than 20 cm. then a bigger field is given.

(4) In Stockholm at the Radium Hemmet :—

The main factor of the treatment is the application of radium by the method too well known to require further description here, but followed by deep X-ray therapy to the parametria.

(5) In Paris there are three main clinics, viz. :—

(1) Regaud, Lacassagne, Goutard.
(2) Roussy, Wickham, Laborde.
(3) Gosset, Monod, Wallon.

The main difference in the French technique of deep X-ray therapy is the longer focal distance of the X-ray tube and the somewhat heavier filtering, which necessitates a much longer treatment, and consequently the necessity of a fractional dose technique. Goutard's technique of X-ray therapy is briefly this : 180-200 k.v. filter 1-2 mm. Cu or Zn, 4-5 ma. in the secondary current 60-80 cm. focal distance.

As a rule large fields are advocated and the treatment is carried out twice a day, each lasting at least one hour. Each treatment in another field, but repeated until the actual dose on one field is just under an H.E.D. (taking 5-8 hours in all). Such a radiation takes from 15-25 days. The treatment is combined with radium.

This variation in X-ray treatment has been tried in several German clinics, and in some of them has been given up. In Würzburg, however, under Gauss, von Neeff carries out a modification of Goutard's technique, and in Heidelberg this method is liked and being carried out with success.

(6) In Brussels, at the Centre Anticancereux Universitaire, under Dr. Murdoch, the Chef du Service, carcinoma of the cervix is being treated differently. Here the good results are considered to be due primarily to the anatomical position of the cervix, which is especially favourable to the application of radium, as well as to the fact that a considerable dose can be safely applied to the mucosa of the vagina and the uterus and uterine muscle.

Deep X-ray methods have also reached a high degree of perfection and excellent results have recently been obtained by X-rays only; probably, however, the best results are obtained by the use of both radium and X-rays.

Brussels technique consists of an " intracavitary " method of applying radium, and a transcutaneous radiation of deep X-rays. This they considered safer than inserting needles into the cervix (which has been tried by Regaud and his associates, and others), because in the latter case the difficulty of disposing the needles correctly is great; also it is difficult exactly to judge the size of the tumour, and there is danger of coalescence of needles, especially when the tumour is destroyed; the needles then come together and produce irregular irradiation and sometimes radio-necrosis.

Technique.—Biopsy. Preparation of patient: 24 hours' rest and watching; the temperature taken, asepsis cared for, and so on.

Radium is only applied when the temperature is normal; if fever continues in spite of the preparation X-rays are given, which sometimes succeeds in cleansing the local lesion, but may cause narrowing of the cervical canal through sclerosis, which makes the later application of radium more difficult.

Longitudinally grooved aluminium tubes are used for the radium, to ensure drainage and so prevent the rise of temperature which follows the application of radium in a septic case. The intra-uterine tube is left in situ for 9 days, 2,877 mg.h. in all. The vaginal tubes are left in place 5 days, or 3,200 mg.h.

Deep X-rays are given two months after the local treatment by Hohlfelder's method, viz., 186 k.v. 0·5 Cu + 1 Al through each of the four portals of entry. 800 R internally are applied and the treatment is spread over eight days.

Telecurie-therapy, viz., radiation from a 4 grm. radium bomb, has been tried, but the results have not been found as satisfactory as those with deep X-ray therapy.

In conclusion, when we summarize the recent advances in deep X-ray therapy in uterine disease, we find :—

(1) Results in the newer technique in the treatment of small interstitial fibromyomata and climacteric hæmorrhages are excellent, with cure in 98% cases; and, further, that the necessary treatment can now be carried out in much shorter time than formerly, namely, in from 35 to 90 minutes, depending on the type of case.

(2) Modern technique in the treatment of carcinoma of cervix and fundus includes deep X-ray therapy, given either by the "one treatment" method, or by the "fractional" method, following the application of radium in carcinoma of cervix and following total hysterectomy in carcinoma of fundus.

(3) Improvement in results is due not only to the newer types of X-ray installations, which make for more homogeneous and stable radiation of a shorter wave-length and of higher penetration, but also to the more general acknowledgment by gynæcologists of the necessity for better care of the patient, both before and after radio-therapy, such as longer hospitalization, with its possibility of aseptic treatment of the local growth and other measures to accelerate metabolism, and so to eliminate the products of degeneration of the tumour—an important factor in the ultimate cure.

The PRESIDENT said that he strongly disagreed with the use of X-rays in the treatment of fibroids. In exceptional cases their use was justified, but as a routine to destroy healthy ovaries for the sake of a diseased uterus was an altogether wrong principle. Fibroids subjected to X-ray treatment often shrank, but never entirely disappeared, and he had on many occasions had to operate on elderly women because fibroids thus shrunk were giving trouble. There was an undoubted relation between small fibroids in an old woman's uterus and carcinoma of the corpus. The ideal treatment of fibroids was myomectomy, and the technique of that operation was now so much improved that very few cases were outside its scope.

Methods of Obstetric Diagnosis and Treatment at the Rotunda Hospital in 1909, compared with 1929.

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So much work has been done on hormone therapy, endometrioma, the toxæmias, disproportion, etc., that I felt it might be of interest to find out why it was necessary for the present writer to relearn the midwifery he acquired when assistant at the Rotunda Hospital in 1908-11, and to strike a new note on many subjects when taking up the position of Master of the Hospital in 1926.

After five years it is possible to review dispassionately the new ideas, to compare them with the old and to endeavour to sum up what is best. Whether this will be satisfactory or not it is impossible to say, but a study of 20,000 labours should be helpful.

In a recent communication [1] it was shown that excellent work could be done in quite unpleasant surroundings. In 1909 there were two labour wards with stone floors, each room was 27 ft. by 18 ft. In this confined area excellent work was done, but when new labour wards were built, although there was no apparent improvement in statistics the increased space afforded greater comfort in teaching and probably improved the lot of the future patients of the students. However, the old system of two labour wards, whereby a fresh one was used every alternate week, had great advantages.

In spite of comments on the cruelty of shaving pubic hair, we practise it. In 1909 the only patients shaved were those requiring Cæsarean section, pubiotomy

or induction; those who were not shaved got on just as well as their shorn sisters of to-day.

One of the most prominent facts that have come before me is the time of fixation of the head in the primigravida. Textbook has copied textbook and with few exceptions we still find: "the head fixes three weeks before labour begins." I had often noted that many primigravidæ had got on very well with their labour in spite of non-fixation and I decided to study the subject. One of my post-graduates, St. John Moffatt, worked for me and a summary of some of his results is as follows:—

(1) Non-fixation of the fœtal head in primigravidæ, commencing labour, is relatively common, and amounted in this series of cases to 19·5 per cent. of all cases in which the head presented.

(2) In 75 per cent. of these cases the patients delivered themselves spontaneously of a live normal child.

(3) The labour is, on the average, slightly longer than where the head is fixed.

(4) In 75 per cent. of these cases no reason for non-fixation of the head can be given. Therefore it must be presumed that the same factor is at fault here as in the case of the normal multipara, i.e., lack of tone in the abdominal muscles or flaccidity of the uterine muscle.

(5) In some 10 per cent. of the remaining 25 per cent. of cases, delivery was effected by forceps, applied when the indications warranted their use, and when the conditions necessary for their application were fulfilled.

(6) The remaining 15 per cent. of cases required more serious operative treatment, chiefly the lower segment Cæsarean section.

(7) Definite diagnosis of coming difficulty can only be made early in labour in a very limited number of cases.

(8) The value of an intelligent understanding of physiological labour combined with observations from palpation, rectal examinations, and the avoidance of vaginal examinations cannot be too strongly stressed in this type of case, where the necessity for interference may arise at any time.

(9) The treatment in these cases consists chiefly of: (*a*) thorough examination to eliminate those cases where operative measures are essential; (*b*) a trial labour for all other cases, being prepared to interfere if and when necessary.

These results are illuminating and may prevent unnecessary diagnosis of disproportion from being made.

Pelvimetry and disproportion.—In 1909 we relied nearly entirely on the Skutsch pelvimeter for diagnosis of what we then called contracted pelvis. The Skutsch pelvimeter seems still to be the most favourable instrument, although Tottenham, Thom and others have produced various modifications. If there is time and if an X-ray plant is available, radiographic pelvimetry gives the best results. Pelvimetry should certainly not be discontinued altogether; it is of great assistance in helping to form a conclusion as to possible fit, but one of the great advances in the treatment of disproportion (as it is now properly called instead of contracted pelvis) is the introduction of the Kerr Muller method of determining how the head will fit.

The great difficulty in adopting the head as the pelvimeter is teaching. It is impossible to allow more than a certain number of post-graduates to examine cases and a method has not been devised whereby this subject can be satisfactorily taught in a mannikin. The writer is endeavouring at present to see how far he can base his diagnosis on his own extern method; this has been published and the results have so far been extremely satisfactory for basing an opinion, not only before labour begins but also when following the course of labour. The value of extern pelvimetry in its relation to disproportion has already been shown by the author and Wentworth Taylor [3]. We found that of "294 cases, there were 83 in which the measurements were approximately normal but in which some dystocia arose, whilst in 212 of these grouped under small measurements 83 were delivered spontaneously,

leaving 129 which required some form of operative interference, i.e., 43·8 per cent." This shows the value of external pelvimetry as a guide to treatment.

It is impossible to say why the number of cases of disproportion has increased so enormously; they are not manufactured at the Rotunda; they are difficult to deal with, even though they are amongst the most interesting abnormalities in obstetrics.

The third stage of labour.—When I was assistant at the Rotunda, the students or nurses who did not control the fundus got more opprobrium than they could sometimes stand. The number of hour-glass contractions was large—hence the number of manual removals was large. Now, control of the fundus is not practised and this is one of the great improvements in technique. An observation is kept on the patient's pulse and on the amount of discharge. After thirty minutes, the placenta has left the uterus in most instances. Theobald, some years ago, suggested that the "Garden of Eden" method of midwifery might be best. The writer agrees that this is true of the third stage, and no ill-result has followed the "no control" method.

THE TOXÆMIAS OF PREGNANCY AND ACCIDENTAL HÆMORRHAGE.

	Accidental hæmorrhage	Eclampsia
1909	6	12
1910	6	7
1929	41	8

These figures speak for themselves. In addition, in 1929 there were twenty-seven severe cases of eclampsism and 629 patients suffering from albuminuria. One of the chief reasons this paper was written was to provoke discussion, and information on this point is urgently needed. What is the explanation? Investigations on the toxæmias have been rampant; suggestions for undertaking new methods of investigation of the enormous material at the Rotunda are urgently solicited.

Prenatal care may account for the discovery of some of the cases of albuminuria, but it does not account for the enormous increase in the number of cases of accidental hæmorrhage; this number should be diminished and yet the severe types continue to arrive—some of them moribund. It was thought that the number of eclamptics would diminish—the number is much the same and the type on the whole is more severe than in 1909, many of the milder types being checked as eclampsism in the pre-natal department.

With the heavy onset of toxæmic cases there arose a new phase in obstetrical warfare, so preparations had to be made to resist the attack. Starvation and elimination are still our main reserves, but three days instead of four is the limit of time nowadays before induction is practised. Too lengthy starvation produces acidosis. Glucose is used, though not to the extent that Titus suggests. We have been unlucky with intravenous glucose in spite of all care and it is given by mouth, rectum and into the tissues of the breasts.

In eclampsia itself, the treatment remains the same, except that venesection is now a routine when the blood-pressure is 160 or over, or where the patient complains of headaches or other signs of cerebral irritation. Calcium gluconate and radiostoleum are now being tried for the albuminuric patient, but so far without success. Some readers may remember a stormy argument between Fothergill and Tweedy about the treatment of accidental hæmorrhage, in which the former advocated palliative and the latter radical ("plugging the vagina") treatment. This was one of the most difficult new pills the writer had to swallow, i.e., to cease plugging the vagina. There is no doubt that there is no need to plug the vagina for accidental hæmorrhage. General practitioners have asked, "But what am I to do?" Although accidental hæmorrhage is much more safely treated in hospital, it can be treated in the private house, provided there are two good nurses (day and night). Submammary saline repeated when necessary, puncture of the membranes, repeated doses of pituitary

extract as well as careful dealing with collapse symptoms will cure in most instances.

Still an explanation is required as to why there have been 98 cases of toxæmic accidental hæmorrhage and 50 cases of the non-toxæmic variety in the first four years of the present mastership.

Induction of labour.—Some critics of the Rotunda—and helpful criticism inspired by knowledge is more than welcome—have been very caustic in their remarks on the number of cases of induction of labour. In 1909 and 1910 there were 17 cases; in 1929 there were 111; why did this increase occur? Of these, 38 were by medical means without ill-effect; 34 were by bougies, of which 22 were for disproportion; 30 were by puncture of the membranes. A study of the reports will show the necessity for all these procedures—eclampsism, eclampsia, accidental hæmorrhage, dead fœtus and hydramnios overtum, all were indications.

The writer must confess himself a complete convert to the old-time method of induction by means of puncture of the membranes; he once believed it bad, but having tried most other methods he finds himself in agreement with the many authors who have lately written papers on the subject including Guttmacher and Douglas [4], Jackson [5], and Fitzgibbon [6], and now uses it altogether at full term. So Scheel's ghost may walk in the Rotunda sure of a hearty reception. The greatest disadvantage is its simplicity whereby unnecessary induction may be done. The increased necessity for induction is accounted for by the increase in conditions requiring it.

Inertia.—Uterine inertia is still a dreadful worry and no pains should be spared to teach the student the necessity for discriminating between the primary condition and when it is due to such secondary causes as disproportion. The writer has found the classification of this abnormality into primary and secondary—with its subdivisions early and late—of great value.

The big change in this condition is the almost complete stoppage of oxytocic drugs in treatment. Many suggested helps have been tried including pituitary extract, thymophysin and ovarian extract, but they are all dangerous, and numerous examples could be given to show this. Watchful expectancy, attention to general hygiene and the ordinary soporifics must be used. In this connection contraction ring dystocia has become very popular, but since all oxytocic drugs have been stopped, contraction ring dystocia has also ceased at the Rotunda, so it might be well for this Section of the Royal Society of Medicine to consider whether it should publish a written opinion on this point. That is, to instruct the present practitioner not to use this class of drug for inertia. This advice is given because although small doses of pituitrin may be given with safety in early labour, yet it requires an extensive experience to know when it is safe and the type of inertia case to which it may be given.

Forceps.—In 1909 and 1910 there were 81 applications in each year; in 1929 there were 137. The latter is an incidence of 6 per cent. There is no doubt that by allowing women to strain with the head on the perineum the incidence can be lowered, but humanity must come into obstetrics. Low forceps may possibly add a danger to the patient but it is so small that it may be nearly neglected, but one of the big things in teaching is to try and make the undergraduate and the post-graduate understand what a low forceps is.

The writer studied and practised the use of the Kielland forceps and he repeats that used as a high forceps, they are extremely dangerous, i.e., when used following the rules laid down by their inventor, and this is borne out by various papers on the subject. If used in other positions in the pelvis there is no objection, but it is better for the obstetrician to use the instrument to which he is most accustomed. The mere word "Kielland" does suggest, however, dangerous meddlesome midwifery, and it is time there were two definite terms: (1) Meddlesome midwifery, i.e.,

meddling at the correct time, and (2) dangerous meddlesome midwifery, i.e., meddling at the wrong time.

Cæsarean section.—Little need be said on this subject. From one Cæsarean in 1909 to forty-three in 1929 is a big step, and yet every one of these cases was carefully considered before operation. The increase in the number of sections overlaps the increase in cases of disproportion. This may be due to the fact that the present mothers were the growing children of the war period; it is a little due to ease in transport. Whatever the explanation may be, it appears as if the peak now reached is the summit, and perhaps in another twenty years the annual number will again be one.

The lower segment operation is a magnificent operation, but it is not new—it was being done at the Rotunda in 1910, with a Pfannenstiel incision. At that time, after the abdomen was opened the vesico-uterine fold was put on the stretch and opened transversely, after which the upper edge of the fold was separated from the uterus and stretched to the upper edge of the wound in the parietal peritoneum, shutting off the peritoneal cavity. The present method is an improvement, and has important advantages. Apart from anything else, it does away with the bogey, "Kill the mother and save the child," for it is safer to save both by this operation than to do a craniotomy. In fact, the writer prefers to do the lower segment section on some occasions even though the child be dead.

Vaginal delivery after section.—In 1909, the writer published a case of natural delivery after Cæsarean section. Nowadays, there is nothing unusual in this. Whether a classical or lower segment operation has been done, vaginal delivery may be allowed in a suitable case, provided the patient is in hospital. From an experience of the cases in four years it seems justifiable to state this, and it does not seem to matter whether catgut or silk has been used. The delivery must take place in a hospital with proper theatre accommodation.

The decubitus of the patient.—In 1909 we were very proud of our beds which would move the patients either with feet or head downwards. Now we know that a prone position does not favour drainage, and that the sitting position, with the body at a right angle to the trunk, is the only one which obtains a straight run for the uterine excretions. This is a small change worthy of very special notice.

Level of the fundus.—The level of the fundus in the puerperium is at present engaging our attention, and the investigation, which has only been in being for a short time, has yielded the following results which may change our textbook ideas on the subject. A communication dealing with several thousand cases will be given later, so that only the cases in one month will now be considered. 175 were noted. In 128 normal cases and 47 abnormal cases the findings were as follows (the difference between the normal and the abnormal labour cases was practically nil so that they are given together). There were on the eighth day of the puerperium :—

At the pubes	32
1 finger's breadth above the pubes	7
2 fingers' ,, ,, ,,	28
3 ,, ,, ,, ,,	54
4 ,, ,, ,, ,,	34
5 ,, ,, ,, ,,	9
6 ,, ,, ,, ,,	10
At the umbilicus	1

The greatest number, 30·8 per cent., therefore were three fingers' breadth above the pubes. So far as can be seen at present, the investigation of some thousand cases will probably confirm the findings in this small number, but the fact that involution was incomplete did not affect the welfare of the patients.

The only other point to be considered in the puerperium is the treatment of sepsis. Here is a great change. In 1909 the intra-uterine douche was the routine.

In 1929 no puerperal douches were given. It is shocking to think that in 1909 the curette was still used also. Apart from intra-uterine douching there is little change in the treatment of sepsis.

At the moment intra-muscular antistreptococcic serum with quinine holds sway, but collosol iodine, novarsenobillon, abdominal fomentations, a little glycerine, an occasional hysterectomy, vaccines and other methods are used. As this paper is chiefly concerned in showing the difference between 1909 and 1929, the detailed treatment of sepsis will not be considered.

New drugs and appliances.—It is wonderful that in 1909 we saved our patients as well as we do in the present day without the following: Pituitary extract and its companions, such as pitocin and thymophysin; camphor in oil; glucose; various spinal anæsthetics; ovarian extracts; Gwathmey anæsthesia; oil and quinine method of inducing labour; various methods of artificial respiration in infants: ergotamine tartrate; and the writer, although he uses most of them, believes that they could all be done without.

At the Rotunda nowadays the use of pituitary extract is nearly limited to the completion of the third stage. It is occasionally used in ½ c.c. doses in helping to start labour. It is not used in the second stage, as it sometimes causes retention of membranes and post-partem hæmorrhage.

Anæsthetics.—The Gwathmey method has a small field of usefulness. Twilight sleep is given in selected cases. The hyoscine method, which seems popular at present, was given a trial, was found definitely dangerous for the infant, and has been discontinued. Chloroform is not used. In an institution where complete supervision of the junior staff cannot be practised, it is dangerous. Anæsthetic deaths are not commented on sufficiently; there were five in a well-known hospital in the past year and no explanation was given. Ether is practically safe. Gas-and-oxygen would be used but for the expense.

Willett's forceps.—We use these occasionally in placenta prævia but when the cervix admits two fingers it is not always simple to apply them. We have used them, however, to assist in the labour of the premature fœtus.

We regret we cannot teach methods for resuscitating infants which require any such accessory as a cumbersome cylinder. We teach now to deal with the case as one of shock and to avoid all violent methods of resuscitation. The Schultze method was in vogue in 1909.

Other changes.—The other big changes during twenty years comprise the institution of pre-natal departments internal and external, the X-ray department which is used to confirm clinical findings and for the ordinary methods of diagnosing; and the new ward dealing with ailments of infants up to one year. The last is very useful, but the greatest care must be taken not to admit infectious cases, when such a department is within the precincts of a maternity hospital.

Teaching is conducted on practically the same plan as formerly, except that residence is compulsory. With the amount of material available, clinical methods can be followed and theoretical lectures are given to impress the knowledge. Something must be done about "cases for students." The number conducted does not matter very much. There should be a minimum of two months' residence in a maternity hospital where the number of cases is large, and the average annual number of cases should be considered. Possibly the conduct of from ten to thirty labours should be required, but notes of cases seen in pregnancy, labour or the puerperium should be submitted. The writer has the greatest respect for the General Medical Council, and especially for the obstetrical members of that Council which has done so much to improve the teaching of midwifery. It is hoped before making too many new recommendations they will seek the advice of a committee of men and women who are engaged in *teaching post-graduates and students in maternity hospitals.* It may be asked why this subject is brought up here. It is ventilated because the

writer has seen the advantages of the recommendations already adopted, especially with regard to the old-time methods of students trying to learn their work without residence and the present system: he has seen efforts to improve matters further, and he can see the difficulties that a Council consisting chiefly of general surgeons, physicians, anatomists, gynæcologists who are not obstetricians, &c., must encounter when legislating about the science and art of obstetrics which has changed so much in two decades. Yet if the question were asked, "If the Rotunda were being rebuilt, would you change the plans much?" the answer would be, "No, very little." Bartholemew Mosse was wise beyond his time and a Rotunda built to-day would be much the same as the present building. "Solamen miseris instituit."

Conclusions.

(1) Midwifery has changed greatly during twenty years.

(2) Good work can be done in a small labour ward, but a large one simplifies teaching.

(3) It is not a general rule that the head fixes three weeks before labour starts in the primigravida. Results of the investigation at the Rotunda are given.

(4) While the head is the best pelvimeter, the difficulty in teaching this method is great.

(5) The fundus of the uterus should not be controlled during the third stage.

(6) Accidental hæmorrhage does not require plugging of the vagina in treatment.

(7) Puncture of the membranes is an old-time procedure which appears to be the best method for induction of labour.

(8) Oxytocic drugs have been stopped in the treatment of inertia.

(9) There is a vast difference between the terms "meddlesome midwifery" and "dangerous meddlesome midwifery."

(10) It is difficult to explain the enormous increase in the incidence of Cæsarean section.

(11) An investigation into the rate of involution in the puerperium is in progress. Preliminary findings are given and are slightly different to the teaching of twenty years ago.

(12) Intra-uterine manipulation in the treatment of sepsis is nearly discontinued.

(13) New drugs are mentioned. Many of them can be done without.

(14) During twenty years, the teaching of students has been improved. It is suggested that a committee of real obstetricians should be consulted by the General Medical Council before promulgating new rules.

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Discussion.—Mr. ALECK BOURNE could not agree with the entire neglect of the fundus uteri during the third stage of labour, inasmuch as it was not possible without a hand on the fundus, to be aware of the collection of a concealed post-partum hæmorrhage, and nearly all cases of post-partum hæmorrhage were concealed at first. By this control of the fundus he did not mean violent kneading, but a gentle grasp which would prevent the fundus filling up with blood without the doctor's knowledge. A second point was the condemnation of pituitary extract by Dr. Solomons. Mr. Bourne maintained that the dangers of pituitary administration depended solely on the dose, and that if only two units were given before the child was born no harm of any kind would follow.

Mr. L. CARNAC RIVETT said that his instructions were to make the discussion as acrimonious as permissible within the walls of this Society. Dr. Solomons, however, had cut the ground from under his feet, as he found he was in complete agreement with practically everything in the paper. He (Mr. Rivett) was not going to let a trifle like that impede him. Dr. Solomons said that in 1901 there were two labour wards. Mr. Rivett did not think it was stated what labour wards there were now, but he thought that in any modern obstetric unit there should be labour wards which could be completely closed for cleaning and fumigating periodically. In regard to fixation of the head in primigravidæ, anyone who had conducted a large ante-natal department was quite aware that the text-book statement that the head fixes at the thirty-seventh week was quite wrong. He entirely agreed with the Master in regard to pelvimetry, but he did not think that X-ray pelvimetry was yet established. He could not understand the Master's difficulty in teaching the use

of the head as a pelvimeter. He, Mr. Rivett, taught a method very similar to that shown on the screen by the Master. In his opinion the fit of the head could be diagnosed in most cases by pressing it into the brim with the palm of the hand and not with the tips of the fingers, and he was in agreement with Mr. Bourne that the tips of the fingers produced a good deal of pain. In a few exceptional cases the Munro-Kerr method was necessary, and in that case an anæsthetic was also necessary. Did the Master really mean that he taught leaving the uterus entirely alone during the third stage of labour? and that he had never seen any ill-effects from this? In a report of the Rotunda Hospital for 1930 Mr. Rivett noticed that there were thirty-two cases of post-partum hæmorrhage. Mr. Rivett thought it was very probable that the increased number of ante-partum hæmorrhage was due to more cases being sent in from a distance; and the large number of cases of albuminuria was due to ante-natal examination. Mr. Rivett had long ago noticed that the fundus was often at a higher level than the textbooks led one to believe, but did the Master hope that we would swallow his statement that the fundus was at the umbilicus on the eighth day in a normal puerperium? He was sorry to hear that no advance had been made in the treatment of sepsis, but he would leave Dr. Oxley to deal with that. In regard to anæsthetics, hyoscine was not only dangerous to the infant, but it pushed the full amnesia occasionally producing mania in the mother. He was sorry to hear the Master say that chloroform was dangerous and was not used at the Rotunda. Was the Master stating that this drug was so dangerous that he would never use it, without having tried? Mr. Rivett was very much in agreement with the remarks on teaching, except that he did not consider the number of cases was anything like sufficient, and to turn a doctor into the world qualified to practise any obstetric operation after having personally delivered ten patients, was in Mr. Rivett's opinion one of the causes of the high maternal mortality. Every student ought to deliver at least thirty cases, and have at least ten obstetric operations, including forceps, version and even embryotomy.

Mr. W. GILLIATT said he was in complete agreement with most of the things that the Master of the Rotunda had described as his normal practice, and particularly with the abolition of chloroform in maternity hospitals and in maternity departments. He could not, however, share his feeling of safety at leaving the fundus uteri without a hand upon it. In such a case the hand should be passive, merely to indicate what was happening to the uterus and not used to irritate the uterus. He considered that there had been two other notable and beneficial changes in practice in the last twenty years—the invention and use of Willett's forceps, and blood transfusion, which in his opinion, should be used in every case of ante- or post-partum hæmorrhage where the hæmoglobin estimation was less than 55% forty-eight hours after the termination of labour.

Dr. W. H. F. OXLEY said it was surprising that there had been so little change in the methods of treatment in the last twenty years. During the thirty years he had been actively engaged in midwifery he had come to rely less and less on interference with instruments and more on the use of the two hands where interference was absolutely necessary. Thus, in the 10,000 cases he recently reported, the forceps rate was only 2·9% and only one Cæsarean section was performed.

He thought that careful guarding without irritation of the uterus was necessary, and was confirmed in this opinion by reading the reports of a hospital on the Continent where after delivery the abdomen was covered by a towel and the uterus neither palpated nor observed. Out of 2,623 cases delivered there were 244 cases of severe post-partum hæmorrhage and 240 of retained membranes, numbers far in excess of those where the English method obtained.

In most cases it would be found best to choose one way out of many good ways of treating a condition and to stick to it.

In spite of the slight change in details of treatment, the outlook on midwifery had altered during the last twenty years, owing to the introduction of ante-natal care, the provision of a large number of trained midwives, and the benefit derived under the National Health Insurance, which enabled a prospective mother to leave work during the last few weeks of pregnancy, and to rest longer than formerly during the puerperium. The preventive outlook, practised to the full, had made an enormous difference, and he was sure that we should not get the best results, whatever method of treatment we applied, until every mother had close personal attention during the whole of pregnancy, parturition, and puerperium.

LADY BARRETT said she had little desire to criticize the paper as she was in agreement with by far the greater part of it. She thought, however, that pituitary extract had a use

in medicine but was a drug which required the greatest care in use toward the end of labour when the uterine muscles might be nearly exhausted. With regard to the use of forceps at the end of labour to save the mother pain, she felt that anæsthesia which relieved the suffering and at the same time allowed labour to be completed more slowly and normally was better treatment. Expense should not stand in the way of the use of any anæsthetic which most safely relieved suffering and allowed more normal completion of labour.

Dr. BETHEL SOLOMONS (in reply) said that he definitely did not believe in control of the fundus, nor did he believe that post-partum hæmorrhage was caused by non-control. On the contrary, he was convinced that with non-control there would be fewer cases of this complication and that hour-glass contraction of the uterus would be a thing of the past. The nomenclature "post-partum hæmorrhage" had different interpretations, so that it was difficult to get reliable statistics.

His extern method of estimating disproportion was not painful, and was the best method he knew. He preached gentleness at the Rotunda, In deciding on treatment of any condition he always tried to bear in mind "The General Practitioner." As he had seen severe accidents and even fatalities following the use of pituitary extract in labour, he was inclined to avoid it, even though it might be useful in infinitesimal doses to assist in starting labour and possibly in the very early stages. The main fact was that it required an extensive experience to settle on the type suitable for its use. Fibrillary contraction of the uterus had already been noted from the Rotunda Hospital. He had given his reasons for his belief in a labour ward being of fair size. The patients were separated by curtains, and those under hypnotics were kept in a different ward. The difficulty in getting patients in Dublin to attend the pre-natal department accounted for the fact that he did not divide patients into two cases, "booked" and "not booked."

The puerperium in the eleven cases in which the uterus was at the level of the umbilicus on the eighth day was normal and the bladder was always emptied in these cases.

As chloroform was killing women in most maternity hospitals he had adopted ether as an anæsthetic at the Rotunda. Gas-and-oxygen might be more suitable, but it was very expensive. He disagreed entirely with the use of the curette in the puerperium. It should never be used and no teacher had any right to allow it to creep into his lectures. He taught the use of Willett's forceps at the Rotunda, but they had very little use. Donors of blood were not always available for the general practitioner.

Section of Physical Medicine.

President—F. G. THOMSON, M.D.

[*November* 20, 1931.]

(MEETING HELD AT THE BRITISH RED CROSS CLINIC FOR RHEUMATISM.)

Catalysis in Relation to Mineral Waters.

By ARNOLD WOODMANSEY, M.Sc.

IT is not necessary here to discuss the function of a catalyst in general terms, beyond saying that it is a substance usually specific in each case, which may enormously accelerate the progress of a chemical reaction, even when present in extremely low concentrations. This definition is somewhat narrow, but will suffice. In terms of physiology the most familiar example of catalysis is the action of enzymes.

The property of catalysis has frequently been attributed to medicinal waters in explanation of their curative effects, without any definite indication of the type of physiological process which may be facilitated by such action. It has been invoked mostly in cases in which the constituents of the waters were in quantity or kind insufficient to warrant otherwise clear conclusions as to why they act.

Experimental data are accumulating around the Harrogate waters, indicating well-marked mass effects of various constituents, but it is generally conceded that a complete explanation of their effects must include the rôle of the catalyst. One example of catalytic activity is studied in this paper.

Various observers have directed their attention upon the catalytic action of mineral waters, or some unidentified constituent of them, in the decomposition of hydrogen peroxide, by which molecular oxygen is produced:

$$2H_2O_2 = 2H_2O + O_2.$$

Billard and Mougeot [1], by the method previously recommended by Glénard, measured this effect at Royat. A known volume of hydrogen peroxide was added to suitable quantities of mineral water from the various springs, and after twenty-four hours' incubation at 37° C. the undecomposed peroxide was determined by titration with standard permanganate. Plain water with hydrogen peroxide provided a control. Positive results were reported, which were not due to the alkalinity of the waters. The force of this last statement lies in the fact that hydrogen peroxide is unstable in alkaline solution.

In a further paper by the same authors [2] the time-function of the reaction was investigated, and it was shown that the time-curve of decomposition of hydrogen peroxide was similar to that obtained when the reaction was promoted by organic catalase. The catalytic activity of the Saratoga water was examined by Baudisch and Davidson [3]. It was found to have a high value and was retained even in the bottled state providing oxygen was excluded. Primarily it depended on the ferrous content which was stated to be probably in the form of complex ions, but independent of carbon dioxide.

Schoeller and Rothe [4], experimenting with Karlsbad water, considered the catalytic action to be due to ferrous and bicarbonate ions. The action of Wiesbaden water on hydrogen peroxide was studied by Fresenius, Eichler and Lederer [5]. Ageing decreased the catalytic power of the water which initially varied with its pH value. Artificial waters containing manganese and iron were effective. The relation between activity and iron and manganese content was further noted by Gaisser [6].

The method adopted for investigating the catalytic effect of various Harrogate waters on the decomposition of hydrogen peroxide was as follows: A 250 c.c. quantity of the water to be examined was heated in the thermostat to 37° C., in

which it remained during the course of the experiment, 10 c.c. of hydrogen peroxide (made by diluting Merck's perhydrol to 10 per cent. with distilled water) were added and shaken. Five minutes were allowed to elapse before the first measurement in order that any initial oxidation might be complete. Thereafter, periodically, 25 c.c. amounts were withdrawn and titrated with permanganate solution. The progress of the reaction could thus be followed.

Control experiments using distilled water and hydrogen peroxide showed it to be unnecessary to make any correction.

Catalytic effect of the Chloride of Iron water.

The analysis of this water has frequently been published, and it is perhaps only necessary here to give the following data, which, in inorganic solutions, are those known to influence the reaction in question.

Iron ...	77·0	parts per million
Manganese	0·66	,, ,,
pH ...	6·2	

The equivalent of hydrogen peroxide decomposed expressed in c.c. tenth normal solution of potassium permanganate.

In 30 minutes	...	...	16·5
In 60 minutes	...	...	30·3
In 4 hours ...	...	...	88·0

A semi-parabolic curve may be drawn from these figures, which however is sufficiently shallow to make the average hourly rate during four hours a convenient index of the catalytic power of the water. The type of curve varies somewhat. In the case of the strong sulphur waters, the rate is slightly accelerated during the course of the reaction, but the same duration of experiment was observed throughout.

In the above the hourly rate is 22·0. This is called the index.

A similar experiment was conducted with Chloride of Iron water which had been boiled for thirty minutes under a reflux condenser, to avoid loss by evaporation. The pH was then 7·95 and the hourly rate was 11·1. In this case we find that the process of boiling reduced the catalytic rate to one-half in spite of the more favourable pH reaction.

When the water was boiled and filtered, but made to the original volume by the addition of distilled water, the index fell to 2·13. After exposure to the atmosphere overnight in an open flask and subsequent removal of the precipitate, a value 13·6 was obtained.

These results show that the Chloride of Iron water when fresh, although its reaction is acid, is most active in catalysing the decomposition of hydrogen peroxide. Exposure to air or boiling largely destroys the power. Physicians agree on the advisability of prescribing a medicinal water in its fresh condition.

Effect of the Old Sulphur water.

Any line of investigation of the Harrogate waters would be incomplete without a reference to this spring on which the reputation of the Spa so largely has rested. It has been the instrument of most of the physiological and pathological researches carried out at Harrogate.

When hydrogen peroxide is added to a sulphur water the sulphide is oxidized with the liberation of free sulphur. The reaction is complete within a minute or two, so the initial permanganate titration was delayed for five minutes after mixing as previously stated. This precipitated sulphur was found to have no appreciable effect in promoting decomposition of peroxide.

The catalytic index for the Old Sulphur water was 14·75, which decreased to 24·8 after boiling.

Other waters were examined in the same way and are reported in the table below.

General table.—The figures represent parts per million. In the first column the value for "total solids" is given, in order that the reader may judge the degree of salination of each.

	Total solids	Iron	Manganese	Index of catalytic power
Iron waters				
Chloride of iron ...	7792	77·0	0·66	22·0
Crescent water ...	8086	16·1	0·9	29·0
Alexandra	3136	29·8	0·08	10·5
Pure chalybeate ...	136	9·2	0·15	4·9
Tewit	432	20·7	0·4	13·5
Johns	190	13·7	0·2	7·5
Kissingen	3448	15·4	0·08	7·4
Sulphur waters				
Old sulphur ...	15380	trace	0·3	14·6
Strong Montpellier ...	9277	trace	0·4	14·8
Mild Montpellier ...	6228	trace	1·0	17·6
No. 36	4090	trace	0·04	11·8
Magnesia	2727	trace	0·25	7·5
Harlow	618	trace	0·005	17·3

It is natural to look for a proportional relationship between the catalytic power and some factor or constituent of the water. The values most likely to afford such a relationship are pH, iron or manganese.

The pH of every sample has not been measured, but it can be stated that owing to the large amount of carbon dioxide present in the fresh conditions they are all less than seven. Such variations as are found are quite inadequate of themselves to explain the large difference in catalytic power. The iron also, taken alone, is insufficient to account for them. Moreover, the small iron content of the sulphur water series renders it improbable that in these waters there is a quantitative relationship depending on iron.

In many of the above cases the manganese appears to have some bearing in so far as the index and manganese content are of the same order. Perhaps the most noteworthy exception is the case of the Harlow water which possesses an index out of all proportion to its iron-manganese content.

At the present stage we are unable to decide what may be the active factor or factors, but in view of the results obtained and having regard to experimental work on the Old Sulphur water to which reference is made later, it is hoped that further study will be made of the metabolic effects of others and particularly the Crescent spring.

Effect of medicinal water plus Catalase.

Catalase is an enzyme occurring in almost all living tissue which rapidly decomposes hydrogen peroxide when brought into contact. This, apparently the only function exercised by it led to an investigation of the effect of catalase and natural water together as "co-enzymes." Mougeot and Aubertot [7] have experimented with the same object on French sources.

The enzyme was prepared from pig's liver by maceration with sand and water. After centrifuging to remove the grosser suspensoids, a little toluene was added, which acts as a preservative and does not inhibit the enzyme. A very dilute filtered solution, freshly made from the stock, was used.

Before proceeding further, certain conditions affecting the action of catalase may be mentioned, which are of importance in interpreting the results.

(i) The optimum pH for catalase is seven, but no attempt was made in any of these experiments to adjust or maintain any particular pH value.

(ii) During its activity the enzyme itself is destroyed according to the equation governing monomolecular reactions. Preliminary experiments were carried out by

adding 1 c.c. catalase to the hydrogen peroxide solution as before. Titrations were made every five minutes. The results are averages of three experiments.

	Permanganate value of the hydrogen peroxide decomposed
In 5 minutes ...	10·75
In next 5 minutes ...	2·00
In next 5 minutes ...	0·67
In next 5 minutes ...	0·00

The rapidly decreasing figures show the catalase is completely destroyed in fifteen minutes. Further quantities of catalase may be added to the same solution and the experiment repeated until all the hydrogen peroxide has been decomposed.

(iii) The presence of salts adversely affects the catalase reaction. Physiological saline (i.e., 0·9 per cent. sodium chloride) was compared with distilled water. To each was added the hydrogen peroxide and catalase as before.

	Distilled water	0·9 per cent. sodium chloride
Average of three experiments ...	12·77 ...	4·43 amount of hydrogen peroxide destroyed

These results show that under these conditions of concentration, &c., the salt inhibits the effect of catalase by 65 per cent.

The activity of catalase under these experimental conditions persisted only fifteen minutes. This duration of time was chosen as the standard in the following series. Temperature and quantities were observed as before.

In every case an experiment consisted of three parallel tests :—

(*a*) Hydrogen peroxide in distilled water to which was added 1 c.c. of the catalase solution. This served to indicate the strength of the catalase.

(*b*) Hydrogen peroxide in the natural water without addition of catalase.

(*c*) Hydrogen peroxide in the natural water plus 1 c.c. of catalase.

In the following table are given the results, each being the average of not less than three and sometimes six experiments, obtained with various waters, calculated to percentages (i.e., the effect of the catalase alone being called 100).

	Catalase in distilled water	Mineral water	Mineral water plus catalase
Chloride of iron ...	100 ...	110 ...	148
Crescent	100 ...	62 ...	259
Pure chalybeate ...	100 ...	37·1 ...	109
Old sulphur well ..	100 ...	30 ...	122
Mild Montpellier ...	100 ...	58 ...	130
Magnesia	100 ...	17·4 ...	124·5
Harlow	100 ...	138 ...	212

The actual value of the figures in the second column relative to that in the first is of no importance as it merely depends on the strength of catalase used. It was not convenient to standardize the latter. Also various strengths were used with the same water and the results averaged.

Now supposing the catalase and the mineral water each acted independently we should expect the sum of figures in column 1 and 2 to equal that in 3. In the majority of cases this figure is less than the sum and we must conclude there is an antagonism either due to the "salt effect" previously discussed, or other unknown factor.

In two cases, however, viz., the Crescent and the Magnesia, the combined effect is greater than the sum of the two separate effects; the action of the organic catalase is augmented. We do not know as yet the whole significance of these facts.

It has recently been suggested by Dr. D. Brown [8], by reason of its effect in promoting protein metabolism that the Old Sulphur water stimulates the xanthine oxidase of the liver. This oxidase is necessary in the oxidation of purins in the body. When purins are oxidized hydrogen peroxide is formed in the process. The function

of the catalase is to remove this hydrogen peroxide which otherwise destroys the xanthine oxidase, and so inhibits the metabolism of purins.

It is here suggested that the mineral water may assist the catalase in its normal function and by more prolonged action thus exert its known influence in the metabolism of protein partly by this mechanism.

SUMMARY.

(1) The Harrogate waters in vitro act as catalysts in promoting the decomposition of hydrogen peroxide.

(2) They are most effective in the fresh condition.

(3) Some inhibit the action of organic catalase but two are definitely favourable in increasing its action.

(4) The Crescent water possesses the highest catalytic index and it is also the most pronounced stimulant of catalase. It is suggested that the metabolic effect of this water might be investigated as an interesting line of research.

(5) Reference is made to an hypothesis by which the catalytic function in the body may be explained.

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CASES.

Spondylitis in Twins.—M. B. RAY, M.D.

The twins are males, aged 31.

(1) F. D. The condition began twelve years ago with pain described as sciatica, followed by progressive stiffness of spine, shoulders and hips.

Present condition.—Syndrome of rhizomélique spondylitis (Marie-Strümpell type). Rigidity accentuated in spine and shoulders, less in hips. Osseous and tendon reflexes increased, especially in upper limbs. Atrophy of pectoral muscles and diminished chest expansion. Skiagram shows lipping of vertebræ.

(2) H. D. First noticed pain in back four years ago. Upper part of spine and right shoulder now involved. No symptoms of involvement of nervous system. Skiagram shows commencing lipping of vertebræ.

Discussion.—Dr. A. P. CAWADIAS said that in these patients the unknown external ætiological factors (no infection has been found) acted on individuals who showed the same constitutional predisposition, being twins. The first result of this interaction of external and of constitutional factors had probably been a disturbance of the regulators of metabolism of bones and joints (endocrines ?, vegetative nervous system ?). The end-result of these disturbances were osteo-arthritic lesions localized in one shoulder and spine in one case, in both shoulders, both hips and spine in the other case, which thus showed the anatomo-clinical picture designated, since Pierre Marie, as rhizomelic spondylitis. To enter into discussions as to the pigeon-holing of the anatomo-clinical lesions meant to lose sight of the real basic problem, namely, the physio-pathology of the condition, and thus to lose sight of the indications of therapeutical handling.

The problem of chronic rheumatism must not be tackled at the periphery, that was to say in the lesions of the joints, which, as he (Dr. Cawadias) had concluded from a great number of post-mortem examinations, were always mixed, and thus did not lend themselves to a hard-and-fast anatomical classification, but at the centre, namely at the metabolic disturbances governing these lesions. These metabolic disturbances were in their turn determined by a constellation of ætiological factors, external as well as constitutional, a constellation which varied for each individual patient, and did not allow of any hard-and-fast ætiological classification.

Dr. C. W. BUCKLEY said that these cases were of great interest as demonstrating the importance of the "soil" factor in arthritic conditions. He deprecated the use of the term "rhizomelic" as being obsolete. There were two principal types of spondylitis with clearly

marked differences in history, symptoms and X-ray appearances, namely, the ankylosing type which belonged to the proliferative or atrophic group of arthritides, and the osteo-arthritic type which was degenerative and occurred in later life. The cases shown were typical examples of spondylitis ankylo-poietica, and it was most important for prognosis and treatment to recognize their essential difference from such a degenerative process as osteo-arthritis. Mixed types might occur but only in much later life.

Dr. KERR PRINGLE thought the condition in H. D. might be connected with his employment as an engineer, his work being mainly concerned with a lathe.

Idiopathic Hydrarthrosis.—F. D. HOWITT, M.D.

A. C., male, aged 61. Engineer.

Duration of disease, seven years. Supposed cause, erysipelas of face seven and a half years ago.

History.—Four months after recovery from erysipelas, pain and swelling developed in knee-joints.

For the first four years the pain and swelling did not interfere with locomotion. Three years ago a small swelling was noticed behind the right knee-joint, which increased in size slowly until it was as large as a turkey's egg. Eighteen months ago a swelling appeared in the left calf and a few months later a smaller appeared directly behind the left knee-joint. These swellings, particularly that behind the right knee-joint, interfered with locomotion.

On examination.—Twelve months ago these swellings were found to be fluid in consistence, and were diagnosed as large Baker's cysts resulting from severe osteo-arthritis of the knee-joints.

A year ago the swellings were operated upon successfully so far as the left calf and right knee-joint are concerned, but the operation on the left knee was a failure, and the swelling recurred within a month.

The patient now complains of great pain in the knees, which interferes considerably with his sleep and work. Both joints are distended with fluid, and the fluid in the swelling behind the left knee can be compressed into the knee-joint.

Wassermann reaction negative.

The patient has taken iodine and salicylates by the mouth. Locally, he has been blistered, and has been given ionization, massage and faradism and radiant heat to the joints and muscles, without avail.

Paralysis Agitans.—C. B. HEALD, M.D.

E. B., male, aged 63, is suffering from typical paralysis agitans, associated with joint pains, distributed mostly over the Parkinsonian area.

This case is not shown from the arthritis point of view, but to call for suggestions for treatment of Parkinson's disease by physical methods, my experience being that ordinary physical methods, other than diathermy, which gives temporary relief, are useless.

He has no experience of X-ray or radium therapy applied either to the epigastrium or to the base of the brain, in these cases.

Discussion.—Mr. P. BAUWENS said that at the Royal Westminster Ophthalmic Hospital he had had occasion to treat an eye condition in a patient with a very advanced Parkinsonian syndrome. The treatment involved the application of diathermy to both eyes, and therefore the current passed through the base of the skull. The improvement in the patient's condition was remarkable.

Dr. E. STOLKIND said that during the last eleven years he had treated a number of cases of paralysis agitans and post-encephalitic Parkinsonism, which, in his opinion, was a form of paralysis agitans of infectious origin. In some of these cases there was pain in the extremities, sometimes in the joints. The pain might be of central origin or neuralgic in character, and might last for many months and pass away, even without treatment. In

almost all his own cases of paralysis agitans the patients were advised to have baths, physical exercises, &c., in addition to taking hyoscine bromide or stramonium. Of late years he had also given intramuscular injections of iodide (sodium, etc.). In some of the cases improvement lasted a considerable time. It was very difficult to judge the influence of treatment in Parkinsonian disease and Parkinsonism, as e.g., the patient, who dragged his legs with difficulty, was able to walk quickly—or even run—at a command or under emotion.

Arthritis Associated with Graves' Disease.—BERTRAM S. NISSÉ, M.D.

Eva M. G., aged 34, married. During last twelve months has had articular pains coincident with swelling in neck. Nothing of importance in past history: transient swelling of neck had been noticed at about age of 15, but this rapidly subsided.

Condition on examination (29.10.30).—Patient nervous and excitable. Fine tremors of outstretched hands; moist skin; tachycardia, high pulse pressure; fullness of neck. Eyes prominent, but no definite exophthalmos. Puffiness of interphalangeal joints of right hand; pain, swelling and limitation of movement in right knee, pain in muscles of right shoulder; movements of other joints free.

General treatment prescribed.—A course of Lugol's iodine, and local applications to the joints. Definite improvement resulted and patient returned to work.

3.7.31.—Definite signs of dysthyroidism. Admitted to hospital, and afterwards sent for convalescent treatment.

16.11.31.—Still shows marked dysthyroidism with definite exophthalmos, tremors, tachycardia, and swelling of neck; continues to lose weight although subjectively she feels better, There is now puffiness of interphalangeal joints; both knees are swollen and painful, left more than right: limitation of movements in both knees and left ankle. Some pain complained of in moving shoulder-joints and left elbow. Radiological examination, negative.

The case is shown as an example of arthritis associated with a disturbance of endocrine equilibrium.

Suggestions are invited for the treatment of this patient. In view of the fact that she has to earn her living and has already been away from work for a considerable period surgical measures are most probably indicated.

Discussion.—Dr. PHILIP ELLMAN said there was little doubt about the correlation of the two conditions. The arthritis came under the metabolic group as opposed to the toxic.

He had noticed that the metabolic group was often associated with hyperpiesis and obesity, thus forming a characteristic syndrome. In this particular group considerable success has been obtained by the use of intrapelvic diathermy.

Dr. W. S. C. COPEMAN suggested that a course of insulin might be useful. He had been trying this in cases of true rheumatoid arthritis, in which there was probably a derangement of the internal gland secretions, and abnormality in carbohydrate metabolism, with considerable success.

Bilateral Osteo-Arthritis of Hips with high blood uric acid.—W. S. C. COPEMAN, M.R.C.P.

Male, aged 43. Married. Increasing stiffness when climbing stairs for last sixteen years. Pain persistent in right hip and knee, greatest in morning; slightly less as day progressed. History of gonorrhœa twenty-two years ago. Treated. No symptoms since. In Army until two years ago. Since commissionaire.

Physical examination.—Much limitation of abduction and rotation in both hips. No signs of renal deficiency. Wassermann reaction negative. Blood uric acid 5·2 mg. per 100 c.c.

Radiological report.—Double osteo-arthritis of hips.

Points of interest are the occurrence of a bilateral osteo-arthritic lesion and the high uric acid in the absence of gouty symptoms or renal disease.

Dr. F. BACH suggested that the "trauma" in these cases which was generally assumed, might in some instances be really a congenital abnormality in the head of the femur.

Classification of Chronic Rheumatic Arthritis by means of Radiograms : Demonstration.

Dr. S. GILBERT SCOTT showed a series of lantern slides demonstrating the lines of investigation that he is at present following at the British Red Cross Clinic for Rheumatism, the object in view being a classification of all chronic rheumatic arthritis into definite groups by means of a radiographic dissection, that is, based upon the pathological changes in the joints, as demonstrated by radiographs. He pointed out that before any such investigations could be undertaken, it was essential that a "standard" radiograph be established. He takes a radiograph of the hand of all patients. This he uses as his "standard" tissue, utilizing at the same time a standard method of technique. In this way it is possible to compare one radiograph with another, the special value of the method being to gauge the increase or decrease of bone calcium.

He considers that there is normally an ebb and flow or circulation of bone calcium. As the characteristic feature of the atrophic or rheumatoid arthritis group is a loss of bone calcium, early detection of a loss of bone salts is important.

Dr. Scott showed examples of the characteristic changes seen in the three main groups : atrophic or rheumatoid arthritis, gout, and osteo-arthritis. The infective arthritic group is still incomplete. The changes seen radiographically are so distinctive that this method of classification must eventually form the basis of differential diagnosis.

Section of Surgery.

President—Mr. C. H. FAGGE, M.S.

[*October* 7, 1931.]

Fibrosarcoma of the Corpus Cavernosum.—ARTHUR EVANS, M.S., H. A. D., male, aged 44.

The patient came complaining of a tumour of the penis (fig. 1). He first noticed a minute swelling, "the size of a pin's head," on the dorsum of the penis nine years ago. This had slowly increased in size.

FIG. 1.—Fibrosarcoma of corpus cavernosum.

Four years ago it was the size of a hen's egg. During the last twelve months it has grown much more rapidly, and now measures thirteen inches in its greatest circumference.

Condition on examination.—When the patient is viewed in the erect position the penis is entirely hidden by the tumour, but on examination it is found that the terminal two inches of the penis is free and is not involved in the growth. There is no trouble with micturition and the emission of semen is normal.

There are dilated veins coursing in the skin which covers the tumour and the skin is dusky red and slightly œdematous, but the changes in the skin cannot be

considered as evidence of the extension of the tumour into the superficial structures, since they can be accounted for by the fact that the patient has painted the tumour with iodine daily for more than a year.

The tumour is smooth, rounded and indistinctly lobulated ; it is in no part cystic, but is tensely elastic, and feels as would a fairly soft fibroma or a fibrosarcoma. The proximal portion of the penis cannot be palpated from the dorsum, being completely covered by the tumour, but examination in the perineum reveals the bulb of the corpus spongiosum to be normal ; the latter can be palpated in its whole length, running on the under-surface of the tumour, and apparently is not involved in the disease. The proximal portions of the corpora cavernosa can

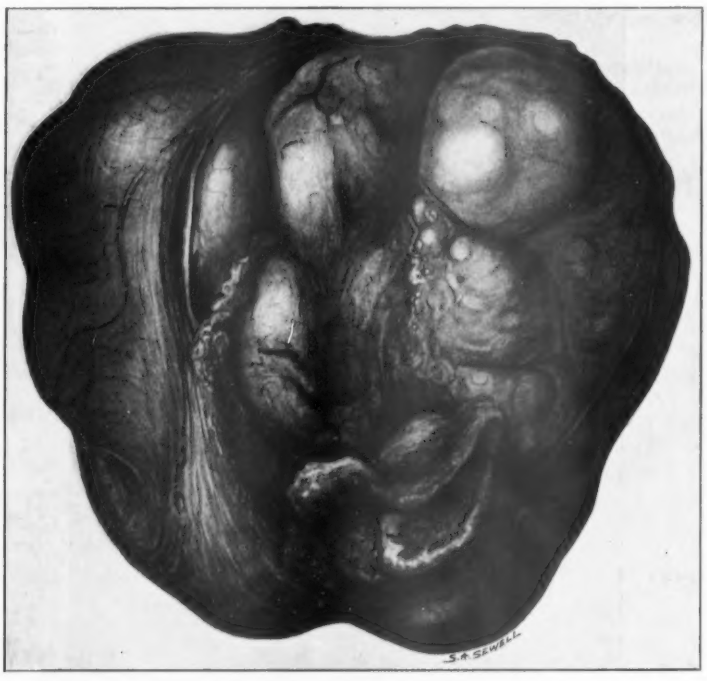


FIG. 2.

also be palpated in the perineum, and these seem to be normal until they are lost in the mass of the tumour.

Seeing that micturition was normal and that, presumably, the corpus spongiosum was free from tumour formation, I decided to try to save this and to remove the tumour, together with as much of the corpora cavernosa as might be found involved in the growth.

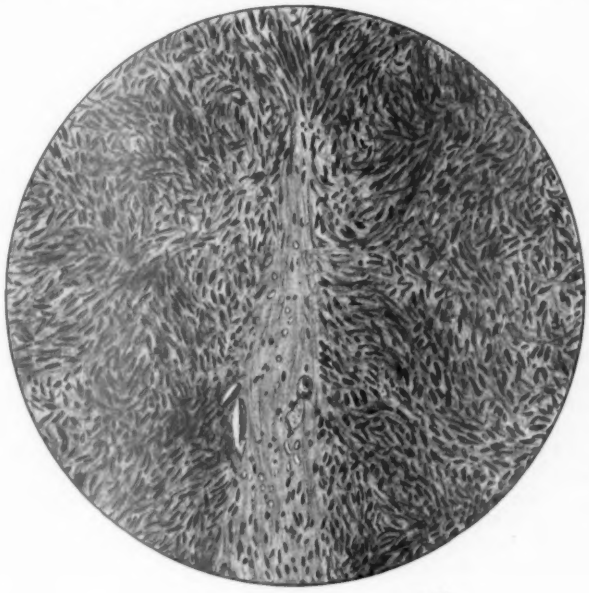
A large gum-elastic catheter was passed into the bladder. Two lateral incisions were made over the upper part of the tumour, the incisions meeting at the symphysis pubis and at the anterior pole of the tumour.

The proximal end of the tumour was exposed, and, keeping close to this, it was easy to enucleate it from its surroundings.

On approaching the body of the penis it was necessary to cut through a dense fibrous sheet which made a common investment of the tumour and the body of the penis, binding them tightly together. When this had been done along both sides the tumour could be separated almost in its entirety from the corpora cavernosa, except that for about one and a half inches it was firmly incorporated with the right corpus cavernosum. This portion of the corpus cavernosum was excised and removed attached to the tumour (fig. 2).

When the wound had been sewn up, the gum-elastic catheter was replaced by a soft rubber catheter and this was removed the next day.

In the course of the operation the dorsal artery and veins of the penis were tied. As a result of this the prepuce soon became very œdematous and had to be slit up



× 80.

FIG. 3.

along the dorsum, and within a few days a narrow circular zone of skin immediately proximal to the glans penis sloughed. The raw area thus produced soon became epithelialized. The patient has remained well; micturition is normal.

Pathological report.—The tumour is coarsely lobulated, and of firm consistence, and measures 4½ by 4 by 2 in. (fig. 2). The section shows it to consist of a firm white tissue intersected with grey semi-transparent areas. A portion of the corpus cavernosum has been removed with the tumour, with which it is intimately connected.

Microscopical examination shows the growth to be a fibrosarcoma (fig. 3), consisting of spindle cells with a varying amount of fibrous tissue, more abundant in the semi-transparent areas. A section through the portion of corpus cavernosum shows that the growth appears to have taken its origin in the fibrous capsule of the corpus cavernosum (fig. 4, page 4).

This specimen is in the Museum of the Royal College of Surgeons (No. 7822.1).

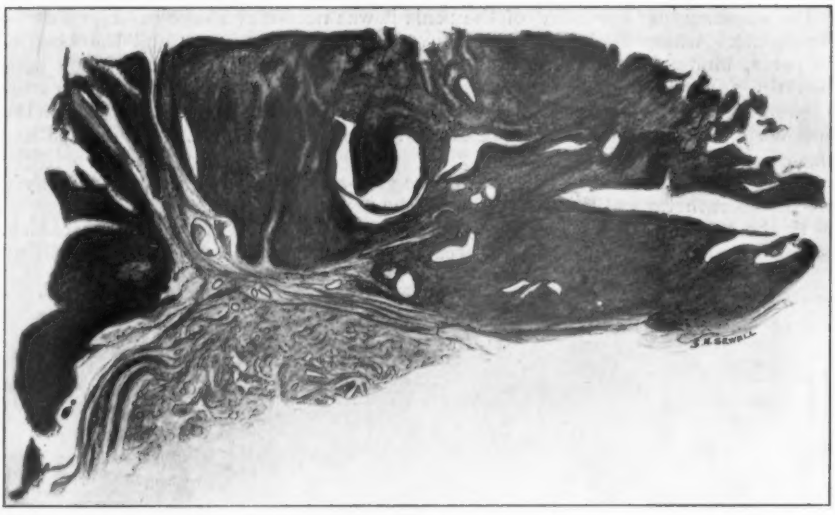
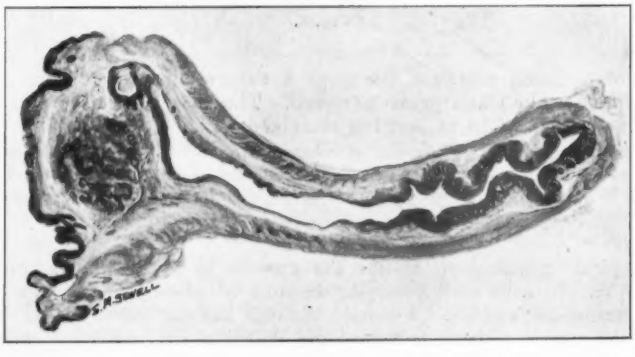


FIG. 4.

Lymphoma of the Vermiform Appendix.—ARTHUR EVANS, M.S.

The patient, a man aged 55, complained of pain in the right lower abdomen, with some tenderness on pressure and thickening in the cæcal region. There was a previous history of constipation and of occasional attacks of vomiting.

Figure 1 shows a section of the vermiform appendix which was removed by operation, together with a small portion of the adjacent cæcum. At the junction with the appendix the wall of the cæcum is thickened so as to form a somewhat rounded swelling, about ½ in. in its chief diameter, which partially obstructs the orifice of the appendix. The swelling is covered with normal mucous membrane. The mucous membrane in the distal half of the appendix is increased in thickness; in the proximal half it is atrophic, and the lumen of the tube is slightly dilated. The muscular coat of the appendix is hypertrophied.



× 2.

FIG. 1.—Lymphoma of vermiform appendix.

× 80.

FIG. 2.

Microscopical examination shows the swelling to consist of masses of lymphoid tissue, bound together by tracts of fibrous tissue. Some of the vessels exhibit marked hyaline degeneration, with narrowing of the lumina. The thickening of the mucosa of the appendix is caused by increase in the lymphoid tissue (fig. 2).

The specimen is in the Museum of the Royal College of Surgeons (No. 6399.2).

Fibromyoma of the Kidney.—ARTHUR EVANS, M.S.

The patient, a woman, aged 36, first felt a slight heaviness in the epigastrium, and burning sensation in the stomach, in 1925; there was "swelling of the stomach," but no flatulence or vomiting, and the symptoms occurred in the form of recurrent attacks lasting about two days. During the next two years the attacks became more prolonged and frequent, and vomiting occurred at times. In 1928 the swelling became constant. At the operation in January, 1929, the tumour was found to have no connection with the pelvic viscera; it was retroperitoneal and its pedicle was attached to the capsule of the left kidney. After removal of the tumour the left kidney was excised and appeared to be normal except for the presence of a small cyst at the upper pole. The patient made a good recovery. She has been seen several times since the operation and her good health is maintained.

Pathological report.—A large oval tumour, measuring 13½ in. in length and 9½ in width, removed by operation from the retroperitoneal region. The surface of the tumour is somewhat roughened, but not lobulated (fig. 1). At one side is a depression from the centre of which radiating lines, resembling bundles of muscular fibres, extend over the surrounding surface. At the lower part of the tumour is a small projection, about ¼ in. in width, which is the divided pedicle, by which the growth was attached to the capsule of the left kidney near its outer border a little below the middle. The section of the tumour shows an external layer of solid tissue, about ¼ in. in thickness on one side of the section, but much

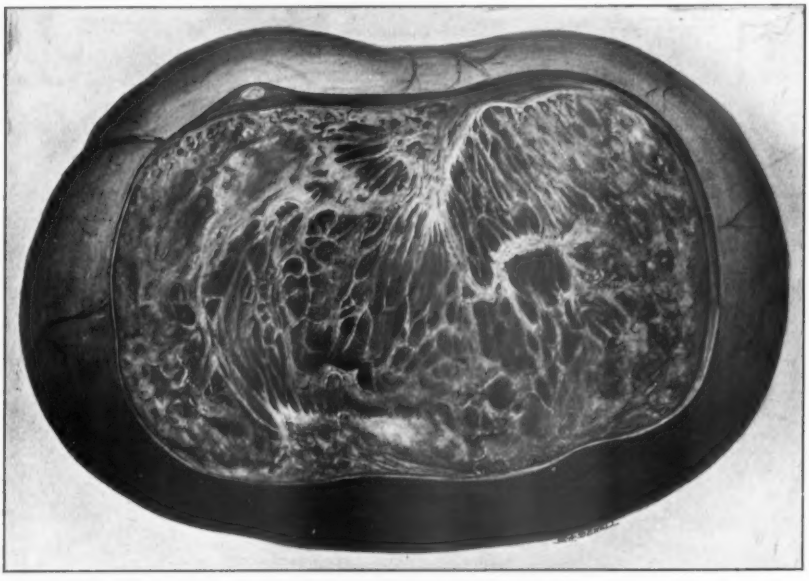


FIG. 1.—Fibromyoma of kidney.

× 100

FIG. 2.

thinner on the opposite side: it is of a pinkish grey colour and at one spot contains a definite nodule, about ½ in. in length. The main central part of the section consists of a coarse honeycomb structure, the spaces of which are filled partly with mucus, partly with a clear fluid. The septa separating the spaces are for the most part extremely thin and transparent, but in the less degenerated places the tissue bears a resemblance to that at the periphery. The tumour weighed 19 lb. and before section was made it had the general characters of a cyst.

Microscopical examination shows the growth to be a fibromyoma (fig. 2), which has undergone extensive liquefactive degeneration (fig. 3).

Before operating I thought the tumour would prove to be an ovarian cyst with a long pedicle. When I found, on opening the abdomen, that the cystic swelling was retroperitoneal I thought I was dealing with a large hydronephrosis; and it

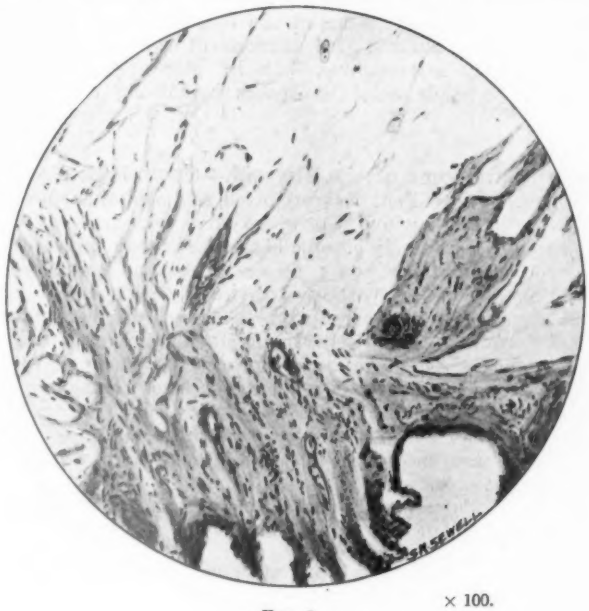


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FIG. 3.

was only when I had freed the tumour from its surroundings that I was able to establish its origin, not from the pelvis, but from the capsule of the kidney. Before the operation, during the operation, and after its removal, the tumour had all the characteristics of a thin-walled cyst, and even in the early stages of its preparation for mounting in the Museum of the Royal College of Surgeons it was regarded as a cyst, until a section across it (fig. 1) revealed its interior structure.

This specimen is in the Museum of the Royal College of Surgeons (No. 7419.5).

Hæmangioma in the Spleen.—CLAUDE FRANKAU, M.S.

The specimen shown is a slice of the spleen forming about one-fifth of the whole tumour. At one top corner is a portion of normal spleen; the great mass of the tumour is somewhat lobulated, showing dark green and greyish areas and some cystic spaces containing yellow necrotic material. The capsule is formed of greatly attenuated normal splenic tissue.

The tumour was removed from a man aged 43, who had noticed a lump for eighteen months; this had gradually increased in size and he only sought advice because of inability to bend forward with comfort and because of some respiratory embarrassment. On examination the tumour was found to extend from the level of the fourth rib to the crest of the ilium and appeared to completely fill the left loin. Blood examination showed a slight secondary anæmia only.

An intravenous pyelogram showed delayed excretion of dye and gross deformity of the pelvis of the kidney: this erroneous finding was probably due to pressure on the renal pelvis.

The tumour presented practically no difficulty in removal: the pedicle was remarkably small, only four ligatures being required for its control. The man made an uninterrupted recovery from the operation.

The tumour, after removal, weighed 8 lb. and measured 10 in. by 8 in. by 5 in. Sections showed considerable dilatation of the blood channels at the union of the normal spleen with the tumour. All sections of the tumour showed extreme degeneration, apparently myxomatous, but in places the ghostly honeycomb arrangement could be made out, the spaces containing red cells. Diagnosis: hæmangioma.

Specimen of Carcinoma of the Thyroid.—CECIL P. G. WAKELEY, F.R.C.S.

Clinical history.—The patient was admitted to hospital as an emergency case, because of difficulty in swallowing. For twenty-five years he had noticed a swelling in his neck and this had gradually become bigger. He was cyanosed and dyspnœic on admission and, within three hours, an operation was performed under intratracheal gas and oxygen. The thyroid was exposed by means of a "collar" incision.

The tumour was fairly well encapsulated, and the lower part was retrosternal; removal was not difficult. Patient recovered well from the operation.

The specimen shows a hemisection of encapsuled tumour of the thyroid. No normal thyroid tissue can be seen on the cut surface. In the upper part of the tumour there is a great amount of fibrous tissue with an attempt at lobulation. Here and there are small cysts, areas of hæmorrhage, and plaques of calcareous deposits. The lower part shows less fibrous tissue, is more vascular, and contains a large central necrotic area.

Microscopically, the upper part of the tumour is adenomatous, and the lower part carcinomatous.

Mr. R. S. PILCHER, M.S., F.R.C.S. (introduced by Mr. J. TAYLOR, F.R.C.S.) showed the following three specimens:—

(I) Spermatocytoma.

Clinical history.—Age 44. Lump for eight years; first appeared after an injury. Tumour attached to scrotum, no hydrocele, mass of glands in abdomen. Primary tumour excised, glands treated by X-rays.

Specimen.—Half testicle with area of skin. Whole organ replaced by tumour. Histology: One type of tissue only, masses of round cells, with deep-staining nuclei in a stroma beset with lymphocytes.

(II) Tuberculous Epididymitis.

Clinical history.—Age 39. Pulmonary tuberculosis. Swelling of testicle ten days. Patient very ill with high fever, tachypnœa and tachycardia. Excision under local anæsthesia.

Specimen.—Caseous tuberculosis of epididymis, miliary tubercles in testis, empyema of tunica vaginalis.

(III) Duodenal Ulcer.

Clinical history.—Female, aged 23. Death from hæmatemesis after a prolonged febrile illness with enlargement of liver and spleen, ulcerative rash, enlargement of glands and tumours in breasts. No diagnosis. Ulcer not suspected.

Specimen.—Duodenal ulcer with maximum diameter of 5 cm. Pancreatico-duodenal artery eroded in floor. Pancreatic and bile-ducts exposed, but not eroded. Recent perforation sealed by adherent liver.

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Mr. W. S. PERRIN, M.Ch.

[*November* 11, 1931.]

PRESIDENT'S ADDRESS.

Some Landmarks in the History of Rectal Surgery.

By W. S. PERRIN, M.Ch.

FROM time immemorial two topics beyond all others have engaged the attention of rectal surgeons—fistula and piles; I have found records of rectal surgery in both these fields in the writings of Hippocrates.

Hippocrates was born in the Island of Cos about 460 B.C.; he claimed a distinguished descent—on his father's side from the god Æsculapius and on his mother's from Hercules. A genealogical table showing his ancestry as far as Aesculapius has been transmitted to us from remote antiquity.[1] He is said to have died at Larissa in Thessaly at the age of 104. Thus he was born during the great days of the Athenian Republic, and may well have seen the building of the Parthenon or have attended Pericles on his deathbed. More than sixty existing writings are attributed to him. They are of unequal merit and few are regarded as genuine. Most, it is said, were composed by his disciples, many of whom bore his name.

On the subject of fistula he wrote[2]: "The frequent exercise of either riding or rowing occasions a contusion of the soft parts surrounding the anus, which, producing an accumulation of blood, is the origin of the fistula. If a tubercle appears it should be opened before matter is formed, that the intestine may not be injured by its coming to suppuration. But if there is already a fistula, the depth of it is to be ascertained by probing it with a stem of garlick. The primæ viæ must be cleansed, and afterwards the following method pursued; twist round a five-fold strand of very slender raw linen some horsehair, that it may not decay; and by means of a pewter probe thrust it into the fistula; at the same time put the second finger of the left hand into the anus, and when it has reached the head of the probe, which ought to be for that purpose a little bent that way, lay hold of one end of the linen and draw the needle down with the other. The remaining part of the treatment consists in tying the two ends of the string close to the anus, and in tightening the knot every day until the fistula is entirely destroyed. Afterwards, when the fistula has sloughed through, a good deal of burnt verdigris mixed with very small bits of sponge is to be thrown up by means of the probe. This dressing is to be repeated every day taking care to wash the parts well every time after the injection. A sponge covered with honey ought to be introduced, in order to prevent a coalition of the parts before they are entirely sound. For, in this way, the fistula being forcibly expanded by the sponge will not fill up and heal unequally, but it will all become whole together. If this method should prove insufficient it is advisable to make use of the knife, for when the fistula does not get eaten through, having first examined it with a sound, cut down as far as it passes."[3]

He also describes the treatment of fistula by means of a tent smeared with an escharotic.

With very little alteration this account of the treatment of fistula and ischio-rectal abscess—for this is obviously the "tubercle" of Hippocrates—might well be included in any modern elementary textbook of surgery. The importance of early incision of an ischio-rectal abscess is recognized, and, although the treatment of

1 The Genuine Works of Hippocrates, translated by Francis Adams, vol. *i*, p. 9 and p. 23.

2 "Doctrine and Practice of Hippocrates." Francis Riollay, 1783.

3 This condensed account of Hippocrates' method of treating fistula is taken bodily from Riollay with a few additions from Francis Adams' translation of the original Greek, which should be consulted by all who wish to know his exact words. Adams' translation is too long to quote, and I have preferred Riollay's digest to a précis of my own. Riollay's account is substantially accurate, and preserves the ancient flavour of the Hippocratic writings.

fistula by ligature—the "*apolinose*"—has fallen of recent times into disuse, it is not so long ago that it was general. Further, Hippocrates recognizes the importance of preventing too early a healing of a fistula which has been laid open, and lastly advises that if ligature is inefficacious, the surgeon should employ the method which is now in vogue and make use of the knife. Hippocrates does not give a very detailed description of how the surgeon is "to make use of the knife"; this is very likely due to his condensed style, for his writings are often so concise as to be obscure. It may well be that his written contributions to surgery were amplified by practical demonstrations, for recent excavations in Cos have revealed an amphitheatre in which it is recorded Hippocrates used to teach. We may with justice regard these remarks of Hippocrates on fistula as one of the landmarks in the history of our subject.

In tracing the treatment of fistula throughout the ages I found myself confronted by a formidable list of authorities—Celsus, Erotian, Aretaeus, Galen, Oribasius, Paulus Coerulianus, Leonides and Paulus Aegineta among the ancient Greeks and Romans alone, apart from the great Arabians such as Avicenna, Albucasis, Haly Abbas, and Rhazes, all invite examination. Subsequent authors are legion; I therefore shall content myself by giving extracts from one or two authors, separated by two or three centuries or, it may be, a thousand years or so, in my endeavour to find a second landmark. In this search it is impossible to disregard Celsus, who has been called the Latin Hippocrates.

Celsus flourished some four hundred years later than Hippocrates, during the reign of Tiberius, which began in 42 B.C., so that, while our ancestors were endeavouring to repel Julius Cæsar's legions, Celsus was writing his "De Medicina" in eight volumes. He appears to have been the most versatile of scientific writers, for, in addition to his comprehensive work on medicine and surgery (the only one of his books which has been preserved entire), he wrote a treatise on military tactics and another on agriculture. The high estimation in which Celsus' works on medicine are held is shown by the fact that so late as 1831 his "De Medicina" was one of the standard books by which, as one of his translators says,[4] "our public Medical Functionaries have very judiciously resolved that the Candidate is to be tried." Celsus gives a more detailed, but no clearer, account of the treatment of fistula than Hippocrates. He advises both the ligature method and the use of the scalpel. In dealing with a complicated fistula he says that "where several sinuses terminating in one orifice are present, the straight sinus is to be opened by the knife. The others which will then appear are to be taken up by the ligature. If the sinus extends so far inwardly that the scalpel cannot reach it with safety a tent must be introduced." It is interesting that the difficulty and danger of operating on a fistula with a high internal opening were appreciated nineteen centuries ago. There is little advance on the teaching of Hippocrates here.

Of all subsequent writers until John Arderne thirteen hundred years later, I shall deal only with Paulus Aegineta,[5] who wrote a comprehensive treatise on surgery in about A.D. 600. At this time, as far as this island is concerned, history seems silent. Britain had been abandoned by Rome, almost every trace of civilization had vanished and, in the words of Riollay, as far as medicine is concerned "the voice of the Monks only was heard in that awful, long Night."

I have chosen Aegineta because he seems to be the last considerable writer of the Græco-Roman school. He lived just 1,000 years after Hippocrates. We have seen how little Celsus had to add to Hippocrates' teaching, and it is of interest that a surgeon 600 years later favours the knife. Aegineta recommends the introduction of a specillum or sound into the fistula, pulling the end out of the anus and cutting down on the track. If the fistula has not penetrated the gut, "we must perforate it

4 "Aur. Cor. Celsus," by Alex. Lee, A.M., 1831.

5 "Paulus Aegineta," translated by Francis Adams, 1846.

violently with the head of the sound, divide all intermediate parts, and take hold of the surrounding parts, which mostly consist of callus, with a staphylagra and cut them all out." He quarrels a little with Leonides, who prefers to introduce a rectal speculum, and insert the probe through the orifice in the rectum, bringing the end out through the opening on the buttock. Paulus complains that the speculum obscures his view and in the end he is compelled to search for the orifice of the fistula in the folds around the anus, and to insert the probe in the usual way. He advises the timid to treat in the manner of Hippocrates with a ligature. He says: "A fistula is incurable that perforates the neck of the bladder, or extends to the joint of the thigh, or to the rectum. A fistula is difficult to cure when it has no orifice, is blind, and has many windings. All the rest are in general easily cured." Evidently fistula was treated with reasonable success in Aegineta's time.

There is some expansion, but it is difficult to find much advance, on the methods of Hippocrates in Aegineta. Britain has been civilized, and has fallen back into savagery, but the treatment of fistula remains fundamentally the same. The views of the great Arabians, of whom some fifteen were translated from the original Arabic into Latin, need not trouble us, for when Amrou, the lieutenant of the Caliph Omar, sacked Alexandria in A.D. 651, the Arabs made full use of the writings in the famous library which had been founded by Ptolemy, from which they constructed their systems of medical practice. They are merely echoes of the ancient Greeks and Latins. Avicenna perhaps deserves mention, as he prefers in the ligature method to use the bristles of a hog rather than a linen thread, "as they will not putrefy."

We now come to John Arderne: he was born in 1307 and probably gained much of his surgical knowledge from these same Arabian surgeons, living, as he did, some 200 years after Albucasis, one of the most eminent of their number.[6] He wrote several works on surgery, which have been collected and edited by Sir D'Arcy Power, to whom I am indebted for the substance of the following account. Since Aegineta the treatment of fistula had fallen into a bad way as far as Western civilization was concerned. For in contrast to Aegineta's opinion that all fistulæ except the most complicated are "in general easily cured," the surgeons of Arderne's time held very different views. Thus William de Salicet (circa 1245), considered the most skilful surgeon of his age, wrote: "When the fistula is complete, it is assuredly so difficult to cure, that it is better and more honourable for the surgeon to give up the case at once."

Then again, Arderne says of his first patient, Sir Adam de Everyngham, that "he made for to ask counsel at all the leeches and chirurgeons that he might find in Gascony, at Bordeaux, at Briggerac, Toulouse and Narbonne, and Peyters, and many other places, and all forsook him for incurable. And, when he came home, he did off all his knightly clothing, and clad him in mourning clothes in purpose of abiding dissolution, his body being nought to him."* However, Arderne cured him "parfitely within half a year, and he led a glad life by thirty year and more, for which cure I gat much honour and loving throughout all England." Arderne's method was an ingenious combination of the ligature or "apolinose" and the knife, and is as follows: A ligature of four strands (the *frænum Cæsaris*) is passed through the fistula by a probe with an eye (the *sequere me*), so that one end of the ligature hangs out of the anus, and the other out of the orifice of the fistula. The ligature is then knotted and tightened by means of a peg (the *wrayste* or *vertile*) fixed into the widest part of the gorget (the *tendiculum*), which is pushed well into the fistula. A grooved director (the *acus rostrata*, or "snowted needle") is passed along the gorget, until the end projects into the rectum. A shield with a central depression

6 "Fistula in Ano by John Arderne." D'Arcy Power, 1910.

* This is not an accurate quotation but rather an adaptation of the original wording of Sir D'Arcy Power's transcript of Arderne's writings.

(the *cochlearia*) is then passed into the rectum, until the grooved director engages in the central depression. A scalpel is passed along the groove in the director, and the fistula is cleanly divided by drawing the scalpel, the director and the shield out of the rectum in a single movement, the ligature coming away at the same time. The purpose of the ligature is to maintain a correct line, while the fistula is being divided, and that of the shield is to prevent injury to the opposite wall of the rectum during division. Arderne was quite prepared to tackle complicated fistula, for he cured "one that was called Thomas Broune, that had fifteen holes, eight on one side and seven on the other, by which wynde went out with egestious odour." Some of these holes were distant from the anus "by the span of a hand-brede of a man." The daily discharge from these holes was "as much as an egg-shell might take." Arderne gives some good practical advice to the contemporary rectal surgeon in the matter of fees. He says: "For the cure of a fistula in ano, when it is curable, ask he competently, of a worthy man and a grete, an hundred mark . . . of less men . . . forty mark; and take not less than one hundred shilling . . . for never in all my life took I less than an hundred shilling for cure of that sickness." Many of his cases were of course of a simple nature, having one or two orifices only.

Arderne's writings constitute a second landmark. He is the first to give a list of the cases he has cured, many of which would tax the powers of any present-day surgeon. Again, he is more accurate in his description of his methods. Lastly, he is the first surgeon to abandon the use of corrosive agents in his endeavour to cure fistula. It is to this probably that much of his success is due. After Arderne there is little to note till the early eighteenth century when Heister of Helmstadt published an excellent work on surgery which was translated into English in 1743, for, as Sir D'Arcy Power observes, the period of activity which marked the twelfth and thirteenth centuries, and was so well illustrated in the fourteenth by John Arderne, was brought to a sudden close by the Black Death which ravaged the world in 1348. "Wars abroad and economic troubles at home seem to have crushed the spirit of the few survivors from the previous generation, and the next period was retrograde."

I have selected Heister, for he seems to have been one of the first, if not the first, of the eighteenth-century surgeons to embody the current practice of his age in a compendious textbook, and so bridge the gap between John Arderne of the fourteenth century and the writers of the nineteenth. The need for such a work as his is shown by the remarks of his translator, who says: "The Translation of the Book before us, which appears to the World, will obviate the Complaint frequently made among the junior Surgeons, and Pupils of the Art in England, viz., that they are in Want of a general System capable of instructing at large One that is a Learner in Surgery, for the Execution of all the Branches of his Profession; and this, till now, might be affirmed with some Justice." Heister makes the same complaint in the preface of his book. He seems to have taken every opportunity of gaining a thorough knowledge of his subject, for, besides studying in all the important Continental centres accessible to him, on being appointed to teach Anatomy and Surgery in the University of Altorf he obtained leave to make a tour of Great Britain, where he was "from Spring to Autumn collecting everything new in the several Branches of Physic." His book is full of many good things which are not very germane to our subject. I cannot help quoting one, when he is recording his experiences while serving with the confederate armies in Flanders. He says: "At the taking of Mons there were more than 5,000 Dutch wounded alone. I had therefore an ample occasion to extend the bounds of my practice, and was obliged to put on that Intrepidity of Mind which Celsus requires as an essential Qualification in a Surgeon, and, for want of which, some, who are in other Respects skilful Operators do frequently miscarry."

He gives a very good account of the different kinds of fistulæ. He classifies

them into complete and incomplete, simple and compound, and blind or occult. He advises injecting a fistula with milk if it is so crooked that a probe cannot follow it, and thus anticipates our more modern practice of injecting with methylene blue or lipiodol. He is a whole-hearted supporter of the knife, and praises that used by the ancient Greeks, a sickle-shaped scalpel—the *syringotomus*. He says he has also cured superficial fistulæ with the falciform knife with an obtuse point, which was used on Louis XIV, and was hence called the *Bistouri Royal*. He quotes from Dionis,[7] who tells us that "the French were so fond and proud of being in the Fashion, when their King Louis XIV had a Fistula, that they boasted of the Disorder as a Point of Honour, and would even undergo the Operation when there was no real Necessity." He thinks these instruments of little use for deep fistulæ, and praises a scalpel with a long, flexible, silver point, invented by the celebrated Bassius of Halle. He is rather contemptuous of those surgeons, among whom most of us are numbered, who use a flexible grooved probe, pass it through the fistula and cut down on the groove with a scalpel. He says, "which method is cried up by the Moderns, as preferable to all others in deep Fistulæ; but, in what it excels them, I know not." In cutting deep and callous fistulæ he recommends that the external incision should be much larger than the internal, that "there may be free access to cleanse and dress to the bottom of the sinus, and it may be advisable to make two incisions in a cross manner, and to extirpate the callous parts—by the scalpel or scissors." Like Celsus he comments on the difficulty of dealing with a fistula with a high internal aperture, and says: "To say the truth, if the internal Orifice of the Fistula is not within reach of the Finger, the Operation of Cutting cannot well be performed without hazarding the Life of the Patient, and, without that Operation there can be but little Hopes of obtaining a Cure; so that Garengeot judiciously advises the Surgeon, in this Case, to refrain from the Knife, which might cause a fatal Hæmorrhage."

One cannot help feeling that Laurence Heister was not far behind the modern surgeon, and could give a good account of himself at our meetings, in spite of the handicap of having had neither antiseptics nor anæsthetics at his disposal.

After Heister's work it seemed difficult to discover anything better, at any rate in England, for a long time. In 1840 Liston published a book on practical surgery. I had hoped to fix as a landmark in the history of rectal surgery the foundation of the Proctological Sub-Section of the Royal Society of Medicine, but my hopes were dashed when I consulted Liston's book. He was unkind enough to say of rectal surgery, "Many attempts have been made to mystify the subject of diseases of this region, and to separate them, in a great measure, from general surgery. But there is no such difficulty as has been supposed in understanding their nature; the principles which should guide their management are simple, and the means, operative or otherwise, are easily enough applied."

On reflection I felt that Liston's criticism shows that, even a hundred years ago, a vague feeling was about that there were difficulties in the practice of rectal surgery separating it in some measure from surgery in general. This feeling was confirmed when I discovered Liston describing an operation, which he is constrained to admit, the proctologist of the day "did very cleverly." The story is as follows. A pig's tail had been inserted into the rectum of a woman of the town by some medical students, whom she had offended. The tail was prepared for the purpose, the bristles cut short, and was introduced with the thick end uppermost; the consequence was, that any attempt to remove it gave rise to the most excruciating pain. Having secured a strong cord to the apex of the queue, the surgeon introduced a hollow tube—a piece of reed well rounded at the end—and removed both together without pain or difficulty. Apart from this Liston has not much to say on rectal surgery.

7 Heister, Vol. II, p. 262.

After consulting many other works, I have chosen Allingham's textbook (1888) to illustrate the practice of the nineteenth century, when rectal surgery had the advantages of anæsthetics and antiseptics. Allingham's résumé of the practice is excellent. He says, "Fistula is, at all events in hospital practice, the most common rectal disease affecting the adult. Of 4,000 cases, taken consecutively without selection at St. Mark's Hospital from the out-patient department, 1,057 were suffering from fistula, and 196 from abscess, of which 151 subsequently became fistulæ. I have recently examined the records of the in-patients at St. Mark's Hospital during several years, and these show that two-thirds of those operated upon were cases of fistula."[8] I doubt whether two-thirds of the cases operated on in St. Mark's Hospital to-day are fistulæ.

It is interesting to speculate as to why fistula has decreased so much of recent years. Probably it is due in part to an earlier recognition of ischio-rectal abscess and appreciation of the need for immediate and thorough incision as recommended by Hippocrates. I am inclined to think, too, that the growth of the fourth estate during the nineteenth century has played an important part by providing a cheap and abundant supply of material convenient for the rectal toilet. We can scarcely realize how incredibly filthy our not very distant ancestors were. It is only on reading that Pepys, in all his nine years of recording his minutest movements, only once mentions having had a bath, and then doubts the wisdom of the proceeding, that it is brought home to us. I cannot but feel that the plight of the mediæval knight in armour, who found himself in the desert, separated by miles from an oasis and by centuries from the morning paper, must have accounted for much of John Arderne's extensive practice.

Allingham gives an excellent account of how fistulæ of all kinds should be treated. In general he favours incision, but does not despise the ligature of Hippocrates, which he revives in a very interesting form. For the linen thread he substitutes an india-rubber band kept taut by a small oval ring, which can be clamped to the rubber by strong forceps. For certain cases he even uses the tent of Celsus, and describes how by the use of a sponge tent and carbolic acid he cured "a gentleman, aged 50, a free liver and very nervous. Cut or tied he would not be. The experience of Louis XIV was nothing to him, and he thought very disparagingly of an art which could do no better than cut people." However, he readily assented to Allingham making trial of "any treatment not very painful" with a successful result. It is curious to read that Allingham insists on an anæsthetic, unless he is convinced that the patient is very strong-minded. One would have thought that by 1888 the value of anæsthesia would have been so firmly established in the lay mind that no question of insisting or not upon its use could have arisen.

With these few extracts from Allingham our study of fistula ceases. Modern practice is almost exclusively concerned with the knife and the malleable grooved director so unfavourably commented upon by Heister, though I fancy that here and there older and less drastic methods are still employed with success.

Leaving the subject of fistula, and turning to that of piles, we find that Hippocrates has again some pregnant remarks to make. According to Riollay, he says "there are different methods of curing this complaint, every one of which consists in destroying the tumefied parts of the veins. This may be done by fire; for that purpose seven or eight little iron rods should be heated at once to a degree of whiteness; the reason for employing so many is that, after having made use of one the patient may not be obliged to wait until the same is heated again; every knob is then to be burned with these rods. Should they happen to be seated high in the intestine, it would then be proper to dilate it with a convenient instrument. The knobs may also be destroyed by pressing and

[8] Allingham, 5th edition, p. 13.

turning them between the fingers. Galls macerated in astringent wine are afterwards applied, or they may be at once cut off, and the parts dressed with burnt flowers of brass spread on an oiled cloth. For the weaker sex one may confine oneself to the application of astringent remedies." Thus Hippocrates shadows forth the clamp and cautery operation, the ligature method, and employs the ung. galli c opio of modern times in a modified form. His method of manipulation, as far as I can ascertain, seems to have fallen into disuse. Villainously painful as his cauterization may appear, it was recommended no longer ago than 1868, when Demarquay strongly advised red-hot cautery as a cure for internal piles.

Celsus' account of the treatment of piles I found too obscure to understand. One point stands out, however, in his description, that he uses needle and thread to secure the most important vessels; thus the modern ligature methods finds its prototype in writings nineteen centuries old. I shall trouble you with no subsequent writers, excepting Heister of the eighteenth century and Allingham of the nineteenth. Heister cannot be passed over, if only on account of his vivid and almost lyrical description of the "Hæmorrhoids cæcæ" or blind piles. It is observable, says Heister, "that the Veins spent upon the Rectum and Anus are sometimes so much distended with Blood, as to be very painful and resemble Tubercles, either like Peas, Grapes, Wall-nuts, or Eggs, and sometimes they are extended longitudinally like Fingers, without discharging any Blood; and these are by Physicians termed Hæmorrhoids cæcæ, or the blind Piles, which they distinguish from other Tubercles of the Anus by their Colour and Resistance to the Touch; Sometimes these distended Veins are soft and flaccid, giving little or no pain, others are tense, painful and inflamed, tormenting the Patient often to such a Degree, that he can neither sit, stand or walk, often fainting with the Extremity of Pain and more afraid than in real Danger of Death." Heister favours an eighteenth-century version of the method of Celsus. The legs are to be held by two strong assistants; the surgeon is then to tie up the bleeding tubercles with a needle and thread, cutting off those parts which are preternaturally distended beyond the ligature, "taking at the same time care to leave a few of the smallest veins open."

Heister does not believe in operating on every case of bleeding piles. He agrees with Hippocrates that the "hæmorrhoidal Flux, if moderate, is healthy, and, ought not to be suppressed, since the redundant and noxious parts of the Blood are hereby discharged from the Body, many of whose Disorders as the Hyp, Melancholy, Madness, Gout, Asthma etc. are hereby prevented or relieved." Without agreeing with these observations in their entirety, the present-day surgeon might consider whether it is always wise to remove the piles of an elderly patient with a high blood-pressure, to save him the inconvenience of slight rectal bleeding.

Allingham in his fifth edition gives an admirable and exhaustive account of almost all the operations and methods of treating piles which, as far as I can discover, have ever been invented. He mentions no less than thirteen, most of which are now obsolete.

(1) Excision with knife or scissors.
(2) The écraseur of Chassaignac, or the wire of Maisonneuve.
(3) The application of various acids and caustic pastes.
(4) The injection of carbolic acid or other caustic or astringent fluids into the body of the pile.
(5) Cauterization "ponctuée" of Demarquay, Mr. Reeves, and others.
(6) Cauterization "linear" of Voillemier.
(7) Removal by the galvanic cautery wire.
(8) Removal by the clamp and scissors, applying the actual cautery to arrest hæmorrhage.
(9) Dilatation of the sphincter muscles.

(10) Removal by means of the screw-crusher.
(11) Whitehead's operation.
(12) Allingham Junior's modified method of performing Whitehead's operation.
(13) Ligature.

Of these methods I will comment on four. The first is the injection of carbolic acid or other astringent substances into the body of the pile. This method Allingham unreservedly condemns. He says he has tried the injection in many cases. The result was generally "much pain, a lengthy treatment, and the result doubtful; certainly not a radical cure." In view of the present popularity of this method, Allingham's observations are of much interest. Demarquay's operation has already been mentioned; Allingham says he is informed that it is much used by native doctors in China and in parts of India—he expresses no opinion as to its merits. The linear cauterization of Voillemier is, as Allingham remarks, probably unique; it consists in making four cauterization lines just inside the anus, before, behind, on the right, and on the left. The substance of the pile, which must be painted with collodion, is not touched. After discussing all these various methods, many of which he had personally tried, Allingham gives an admirable account of the treatment of internal piles by ligature and pronounces himself wholeheartedly in its favour. He gives the credit of its introduction into surgery to Salmon, fifty years before the date of his writing, viz. 1888. He says that at St. Mark's Hospital there had only been one fatal case in forty years of operating—one out of a total of 670. He cites many other authorities, who report equally favourably, including Mr. Curling, who had only three deaths in more than two thousand operations. Later years have justified the good opinion formed by Allingham of this method, for, with minor variations, it is probably that most practised by surgeons of to-day. With William Allingham I close my little research.

What landmarks then have we discovered in this brief survey? First, the works of Hippocrates, which represent a still older practice. It is difficult to find a second landmark in the eighteen hundred years that elapsed between the writings of Hippocrates and those of John Arderne. All the intervening authors seem to have copied or merely commented on Hippocrates and each other. Indeed at the end of my examination of numerous writers of this period the truth of Bacon's remark was forcibly brought home to me as it was to Riollay: "He that surveys with diligence all the varieties of books shall everywhere find infinite repetitions of the same matter, for manner of delivery divers, but for invention stale and pre-occupate. Thus what at first seemed numerous, after examination is found much abated." We may claim, however, that John Arderne is the second landmark. Living seven centuries after Aegineta, when the science of rectal surgery had fallen into decay, with the crudest instruments and in the worst of conditions he revived rectal surgery, making cures of which modern surgeons might be proud.

From this time knowledge accumulated rapidly. The anatomy of Vesalius in the first half of the sixteenth century, the formation of the Royal Society in the latter half of the seventeenth century, and the consequent researches in chemistry and physics produced the weapons which have made modern surgery possible. Of these the greatest are the introduction of anæsthesia by Hickman in 1823, and of antiseptics by Lister. These last are not only landmarks for us but also for surgery in general.

Despite these discoveries, however, do our methods in the treatment of piles differ essentially from those of Hippocrates and Celsus? Our radical treatment—the ligature—is described by Celsus, and our palliative treatment—galls—was used by Hippocrates. We use the knife for fistula as advised by Hippocrates, and I find that Mr. Miles, in his recent and important work on fistula, recommends that a submucous fistula with a high internal opening should be treated by ligature.

This survey began with a quotation from Hippocrates, and I feel that it should end so. The first of his aphorisms deals with the difficulties of the art of medicine and surgery, and of the principles of the conduct of a case. Though applicable to all branches of the profession alike, it is perhaps the rectal surgeon who will appreciate it most. Hippocrates says: "Life is short, the art is long, opportunity fleeting, experience uncertain, judgment difficult. It is not enough to do what the case requires. The patient, his friends, and the world must besides be attended to."

Section of Therapeutics and Pharmacology.

President—Dr. E. P. POULTON, F.R.C.P.

[*October* 13, 1931.]

PRESIDENT'S ADDRESS.

Obesity.

By E. P. POULTON, M.D.

THE advantages of choosing a subject like obesity for a presidential address are that the disease is very common and of general interest; its origin is still obscure, but it is important from the therapeutic point of view.

Standards of comparison.—In order to determine whether a person is too fat, it is necessary to find his "predicted weight." This is usually deduced from tables relating the age and height with the weight of individuals who have been passed as first-class lives by insurance offices. I have to thank Dr. Otto May for allowing me to use his table compiled for the Prudential Assurance Company from American statistics [1]. It must be remembered that life insurance subjects are weighed and measured in ordinary indoor clothes with shoes, and as my patients were weighed and measured without clothes, I have calculated the predicted naked weight by assuming the weight of the clothes of the insured patients in the table as equivalent to one twenty-fourth of the gross weight for women and one-eighteenth for men, and I have subtracted one inch from the height to allow for the shoes. It is open to question whether on physiological principles, age should be admitted as a factor. Is it permissible to expect that a normal 5½ ft. man or woman should increase in weight by a stone or 19 lb. between 20 and 50 years old? There are many active men and women of 50 years who do not require any such extra allowance, weighing no more than when they were young. Is it not possible that such people may be looked on askance by insurance offices because they are too thin? I have always myself used Ainley Walker's figures relating to stem length and body weight [2]. The advantage of these figures is that it is possible to work out from the standard deviation the range of weight within which about 90% of individuals of any given stem length fall [3] but unfortunately, in practice the range is too big, because, presumably, the length of the legs is not taken into account. There is the man with a rather short stem length who may appear to be too heavy just because his legs are long, and *vice versa*. In this connection Miss Wilcox made some measurements of twenty-three normal women students in the Massage Department of Guy's Hospital; in fourteen of these when the weight was either too big, or average, or too small, for the stem length, the length of the legs was respectively too short, average, or too long, which suggests that a more accurate prediction of the normal weight would be obtained by taking both stem

length and height into account. These figures have been determined for healthy men by Flack [4].

Professor M. Greenwood has very kindly allowed me to give here his regression equation calculated from Flack's Data A (1238 observations), relating Body Weight (kg.) and Height and Stem Length (cm.), in members of the Air Force.

$$\text{Wt.} = 0{\cdot}408 \times \text{Ht.} + 0{\cdot}693 \times \text{S.L.} + 70{\cdot}213.$$

From this formula the predicted weight can be readily calculated if the height and stem length are known. Since the standard deviation is 6 kg. with a mean value of 63·4 kg. it follows that all normal men above the age of 16 (the formula is not applicable below this age) will probably fall within a range of 20% above and below the predicted weight. To be more precise 96% of normals will probably fall within a range of 18·9% above and below. It is noteworthy that this range of normal values is very close to the range of Ainley Walker's normals, in spite of the fact that he was only dealing with two variables—weight and stem length. I should have expected it would have been smaller.

It is interesting that independently and, at about the same time, von Pirquet [5] arrived at the same conclusion as Ainley Walker, comparing the body length with the cube root of the weight, a relation which he designated by the name of "gelidusi." Dreyer [6] has used the circumference of the chest as well as the stem length in predicting the body weight. But unfortunately when people become either very fat or very thin the circumference of the chest becomes unduly increased or diminished owing to the presence or absence of fat, and so this method is unreliable. It is possible that in future more accurate results may be obtained by taking into account the type of the individual as Pende has done? [7]. I should like at this point to make a protest against workers on obesity who only publish the weights of their subjects, without adding a single dimension by means of which the outside person can tell whether they really are too fat or compare one subject with another.

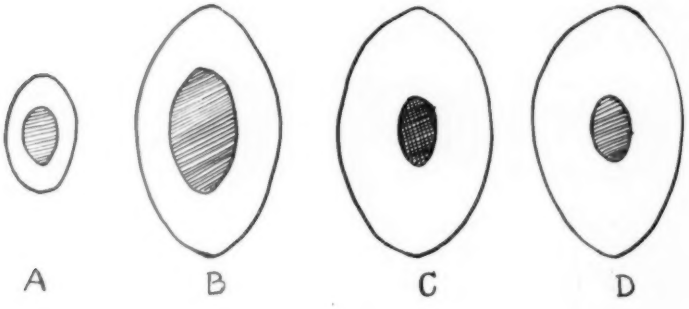
Preservation of normal body weight.—It has often been considered remarkable that most people should keep the same weight from year to year, which means that the amount of food taken in is exactly balanced by the amount burnt in the body. There is general agreement as to the three processes responsible for the burning. (1) There is the so-called basal metabolism of the individual—the respiratory exchange, determined while the subject is at rest in the early morning, before breakfast, so that about fifteen hours have elapsed since the last meal was taken on the night before; (2) there is the specific dynamic action of food stuffs, which means that when food is taken its metabolism in the body leads to increased burning; (3) there is exercise, which increases combustion to supply the extra energy required. It has been suggested that the amount of food taken, which must be equivalent to these three processes, is determined merely by appetite, and that if, while retaining his usual habits, the individual took as an addition to his normal diet a pat of butter or a small glass of milk each day, his weight would increase in a steadily progressive manner. I find it difficult to imagine that such would be the case. Alternately, we have the theory of luxus consumption, which has been discussed by Grafe especially [8]; this means that as the food intake and the body-weight increase, the metabolism increases *pari passu*. It is thoroughly well established that the basal metabolism diminishes during starvation or when the intake of food is diminished, and it is difficult to imagine that such a process would suddenly stop above a certain weight, but the actual experimental findings on this subject in man are not convincing. However, Eckstein and Grafe [9] did show that luxus consumption took place in the dog when the animal put on weight with extra feeding and that this extra burning was inhibited after removal of the thyroid gland. In the

next section I hope to show that in fat individuals luxus consumption does commonly occur in spite of their increase in weight.

There is at present some doubt as to whether in obesity the specific dynamic action of protein and fat is altered. But the observations of Goldblatt, Gardiner-Hill and Forest Smith [10] indicate that after giving sugar there is in some fat subjects a normal increase of the respiratory exchange during the first two hours after the meal; but in other cases giving sugar actually lowered the metabolism—the specific dynamic action was negative.

Exercise in obesity tends to a larger respiratory exchange than normal; the fat person does not work so efficiently, especially if the climate is hot and moist. It is a matter of everyday observation that fat people tend to be sluggish in their habits, and so this factor is less important than usual.

Luxus consumption in obesity.—Obesity is commonly classified as exogenous when it is due to too much food and too little exercise, or as endogenous when it is due to more obscure constitutional factors; further, it is sometimes stated that in exogenous cases the basal metabolism is high, while in endogenous cases it is low and due to hypothyroidism. I am not certain myself that this separation into two types is really very valuable, and I am quite certain that the separation into high and low basal metabolisms is incorrect. The theory of luxus consumption in obesity that Dr. E. C. Warner and I have together elaborated depends on the fact that in many cases the basal metabolism is normal when related to the body surface—a fact that has often been emphasized. The argument can be best illustrated by diagrams (see figure below): *A* represents a normal organism; the shaded part represents the



muscles and glands which produce the heat and the clear part the surrounding adipose tissue. *B* is a similar organism of larger size with the muscles and glands and the fat increased proportionately. The heat produced is proportional to the surface and the extra heat in *B* is due to the increase in the muscle and gland tissue. This is not the case in the obese organism *C*, since the extra size is entirely due to fat while the amount of muscle and gland tissue remains the same as in *A*. If, now, the output of heat in *C* is found to be proportional to the body surface, i.e., is equal to that of *A*, the only possible explanation is that the muscles and glands are working at higher pressure than in *A* because the metabolism of fatty tissue is generally considered to be very low indeed; this means luxus consumption on the part of the muscles and glands, and arguing from Eckstein and Grafe, it may be considered very probable that this luxus consumption is due to over-activity on the part of the thyroid gland. In many fat people where the basal metabolism referred to the body surface is below normal as in *D*, hypothyroidism has been diagnosed, but this does not necessarily follow at all. In *D* the glandular and muscular tissues are working just as hard as

in *A* ; but because the surface is so large the metabolism on calculation is found to be low, and such individuals might be expected to show some diminution in the surface temperature of their bodies. It is reasonable to imagine that if the thyroid is over-active, as is the case with *C*, it may show some wear and tear in the course of years. On the theory here presented hypothyroidism may follow secondarily, in obesity ; but it is not the primary cause. In the same way patients who have had Graves' disease sometimes become myxœdematous in the end. A case of adiposis dolorosa described by Dr. Gullan shows that hypothyroidism does occur in obesity. The patient was a woman, aged 50, who had become stouter as each of her seven children was born, and stouter still after the menopause. Her weight was 286 lb. (20 st. 5 lb.), and she complained of painful sensations in her lower abdomen and thighs ; the facies dolorosa was typical. The pulse was 88, blood-pressure normal ; there was some bronzing of the face, neck, axillæ and nipples. Her weight was steadily reduced to 208 lb., but she was otherwise unimproved, and post-mortem the thyroid gland was found to be small and fibrous, while the pituitary body and the sella turcica were normal [11]. An argument against hypothyroidism being the primary cause of obesity is that there is a marked clinical difference between myxœdema and obesity ; the facies in myxœdema with its puffiness and fine wrinkling is unmistakable, and myxœdematous subjects are often not particularly fat.

In Table I, I have arranged in order of age the cases of obesity studied by myself

TABLE I.—OBESITY.

No	Pt.	Age	Sex	Predicted wt. in lb.			Actual wt. in lb.	B. M. fd.	Comparison %		Remarks	Blood-pressure
				Stem length Av.	Stem length Max.	Life Assur.			DuB.	H. B.		
1	Ro.	29	F.	136	167	138	175	1370	−18·5	−14	Tired. Memory. Cold. Breathless	128/88
2	Go.	32	F.	125	153	127	167	1378	−12	−10	Hair out. Cold. Angeio-neurotic œdema.	118/75
3	Co.	36	F.	131	160	142	164	1408	−10	−6	Hair out. Memory. Cold.	110
4	Go.	36	F.	125	156	130	199	1320	−22	−20	History, 14 years. Fibrositis.	116/50
5	Ch.	38	M.	133	169	153	201	1555	−19·5	−19	Sleepy. Hair out.	134/84
6	Im.	39	F.	101	124	124	152	1115	−25	−21·5	History, 28 years. Takes much salt.	122/87
7	Sa.	40	F.	145	179	149	175	1460	−12	−5·5	Memory. Tired. Cold. Sweats.	138/95
8	Ga.	41	F.	136	167½	149	168	1385	−15	−8		
9	Gr.	44	F.	140	175	137	188	1520	−7	−3	History, 2 to 3 years.	112/90
10	Hi.	46	F.	131	160	136	163	1362	−10	−5·5	Myocardial disease.	120/175
11	Wi.	46	M.	120	150	157	224	1955	−1·5	−3·5	Hair out. Sweats.	215
12	Ra.	46	F.	116	142	130	145	1229	−14	−9	Memory. Hair out. Feels cold and heat.	122/88
13	Fl.	47	F.	155	190	140	186	1425	−13	−8	Memory. Cold. Tired.	165/90
14	McF.	47	F.	116	142	128	189	1490	+7	+13	Cold.	129/92
15	Je.	50	M.	136	183	162	202	1590	−16	−15	Slow. Tired. Memory.	162/102
16	K-W.	50	F.	131	167	144	141	1560	+8	+16·5	Memory. Sweats. Oöphorectomy.	135/100
17	Ke.	64	M.	145	180	153	162	1461	−10	−2·5	Memory. Mild diabetes.	216/125
18	Co.	72	F.	131	160	129	156	1238	−11	−3·5	Cold. Diabetes.	210/120

The average and maximum weight predicted from Ainley Walker's figures and the weight predicted from the life insurance table are given in columns 5, 6 and 7, the actual weight and basal metabolism in columns 8 and 9. The predicted basal metabolism has been calculated both from DuBois' height-weight formula [12], and from Harris and Benedict's Prediction Table [13], and the differences between the actual metabolism and these two predicted metabolisms expressed as percentages of the latter are given in columns 10 and 11. The Harris Benedict figures are a little lower than the DuBois figures ; the DuBois figures are those most commonly used.

and these will be quoted in the text. The normal range is regarded as lying between + 10% and − 10%. In the case of children the body surface has been calculated from the height-weight formula and the calories per square metre per twenty-four hours have been obtained from Charts 13 and 14 of Talbot's paper [14]. In the remarks a poor memory or feelings of cold are indicated by single words. In most of my cases the oxygen alone has been determined by means of the closed circuit apparatus described by Spurrell, Warner and myself [15]; the determinations were carried out over periods varying in length from 15 to 25 minutes or more, the readings being taken every 2½ or 5 minutes, and constant values were obtained in all the cases. In Table I the metabolism of seven cases fell within the normal limits, viz. + 10% to − 10%; the heaviest patient No. 11 with a metabolism of − 1·5% was a naval captain, and of the whole series he was the clearest case of exogenous obesity. In nine cases of the series the metabolism was between −10% - 20%, and in two cases below − 20%. In some of the cases with a low metabolism the symptoms were not compatible with the ordinarily accepted view that hypothyroidism is the cause of the obesity. No. 6 with a metabolism of − 25% was an Indian lady who had no symptoms at all apart from palpitations and dyspepsia. No. 4 (− 22%) who had been fat for fourteen years only complained of sciatica and lumbago. No. 7 (− 12%) complained of feeling the cold; but also sweated a great deal, the latter symptom being inconsistent with hypothyroidism. No. 12 (− 14%), who had had Graves' disease twelve years previously, though she complained that her memory was bad and that her hair was coming out and also complained of both the cold and the heat—another inconsistency. Table II contains a series of eleven pituitary cases, in four of which there was no obesity. In five of the seven obese cases the metabolism was normal. All the cases except No. 3 and 11 were investigated by Dr. E. C. Warner, who kindly allows me to quote his figures. In Table II I have also arranged cases of clinical myxœdema

TABLE II.—PITUITARY CASES.

No.	Pt.	Age	Sex	Predicted wt. in lb. Stem length Av.	Stem length Max.	Life Assur.	Actual wt. lb.	BM. fd.	Comparisons % DuB.	Comparisons % H.B.	Remarks
1×	Ri.	9	M.	101	125	...	105	1180	−13*	...	Hypopituitarism
2×	Mi.	12	M.	...	...	108	116	1152	−17*	...	Hypopituitarism
3	Lo.	13	F.	122	149	116	147	1390	−16*	...	Precocity
4	Br.	15	M.	109	135	136	181	2300	+ 6	...	Hypopituitarism
5	Da.	19	F.	125	153	112	163	1537	− 4	− 1	Hypopituitarism
6	Su.	23	F.	129	159	124	212	1888	+ 6	+ 7	Hypopituitarism
7×	Wa.	27	F.	77	99	...	65	849	−12	...	Ant. lobe deficiency. Partial thyroidectomy
8	Ha.	27	F.	115	141	126	214	652	−13	−11	Hypopituitarism
9	Tu.	35	F.	166	204	149	302	2100	− 1	...	Hypopituitarism
10×	Wo.	35	F.	139	171	146	213	1910	+ 4	+10	Suprapituitary tumour
11	Ni.	37	M.	168	209	174	222	2135	+ 5·5	0	Hypopituitarism
							MYXŒDEMA AND HYPOTHYROIDISM.				
1	Wo.	8	F.	...	...	...	34	370	−35*	...	...
2×	Ho.	32	M.	109	135	120	101	1096	−17·5	−11·5	Myxœdema
3	La.	42	M.	144	179	150	172	1205	−31	−30	Myxœdema
4×	Ma.	48	M.	...	...	147	139	1000	−37	−31	Myxœdema
5×	We.	51	F.	91	117	129	122	898	−29	−27	Myxœdema
6×	Ha.	53	F.	149	183	146	154	968	−35	−30	Myxœdema
7×	Bu.	56	F.	109	133	135	125	990	−24	−19	Old Graves' disease
8	Wa.	66	M.	154	189	153	170	1350	−18	−11	Hypothyroid, mild diabetes

* Talbot standard. × No obesity.

and hypothyroidism. Here again all except Nos. 5, 7 and 8 were investigated by Dr. E. C. Warner. Of the eight cases only three can be looked upon as at all obese In all of them the basal metabolism was well below normal.

Three of my cases have been examined several times, and I give the following details which have a bearing on treatment.

La. was a girl, aged 23, who weighed 126 lb., the average weight from the stem length was 128. An adenoma of the thyroid gland was removed eleven months previously; it was found to measure 3 by 2 in. When I saw her she complained that she felt the cold and that her intellect was dull. The basal metabolism was − 35%. Four months later, after taking 1·2 mg. of thyroxin a day by mouth, the value was − 11%, and she was improved though she still felt cold. Two years later after taking thyroxin most of the time the value was + 3·5%. At this time she suffered from manic excitement with schizophrenic features; her mental condition has recently improved. An interesting point about this case was that though there was evidence of great thyroid insufficiency, and she certainly was not fat, there was no resemblance at all to the ordinary myxœdematous case; she looked a thoroughly normal healthy girl. Is it possible that in myxœdema there is something wrong in addition to a deficiency of thyroxin?

B. W., a woman aged 54, weighed 130 lb.; the average from the stem length was 119, and from the life insurance tables 129 lb.; she complained of feeling cold, loss of memory, palpitation and chronic rheumatism, and her hair was inclined to come out and she was short of breath. The basal metabolism was − 22%. Three months later after taking 1·2 mg. of thyroxin a day by mouth she said she had not felt so well for two years. Her basal metabolism was − 4·5%. One year later she had had no thyroxin for eight months and she was still well; the basal metabolism was − 7%. Here is a case of mild myxœdema which improved with thyroxin, and the improvement was maintained for some months after the omission of the drug. Did the thyroid regain some of its former function?

Ro., a woman aged 60, weighed 149 lb.; the average from the stem length was 102; from the life insurance table 127. She complained of being tired, had a bad memory and suffered from chronic rheumatism. The basal metabolism was − 29%. Four months later with 0·6 mg. of thyroxin a day by mouth she was better, and the basal metabolism was − 12%. Two years later, after omitting thyroxin for a year, she complained of feeling cold and tired, and the metabolism was − 21; so she had relapsed. In the interval she had had an ischio-rectal abscess of tuberculous origin.

I would urge that thyroxin should not be given to obese patients when the basal metabolism is normal. On the theory just put forward hyperthyroidism is already present in these patients and so the tissues are working harder than usual and producing more heat; the addition of thyroid cannot be expected to cause a further increase in the metabolism of such cases. When the basal metabolism is low the matter is different, the thyroid has lost the power to react to the increased stimulus, and so extra thyroid may be given with marked improvement in the symptoms; but it is noteworthy that in spite of this improvement and the corresponding increase in the metabolism the patients do not lose much weight, if any, unless dietetic measures are also taken. Their feeling of well-being must stimulate their appetites.

On the theory that has been brought forward, the surface temperature of the skin should remain constant when the basal metabolism is constant and become lower when the basal metabolism falls. Some years ago Dr. E. C. Warner began making observations on the surface temperature when he carried out basal metabolism tests, using a flat-bulbed mercurial thermometer, and I have followed him in this. Our results are grouped together in Table III. It will be observed that while the temperature of the chest, which is taken just below the right clavicle, and the temperature of the abdomen remain very constant, in spite of the variations in metabolism, the surface temperature of the hand becomes lower as the metabolism falls. Presumably the amount of clothing is normally adjusted to keep the temperature of the body-surface (apart from the hands) constant, as Rubner has suggested.

It might be objected that in obese people there is uncertainty about the calculation of the body-surface from the height and weight alone. In three obese female patients the body-surface has been calculated from the more accurate linear formula of DuBois and DuBois [12], which is obtained from measurements of different parts of the body; this has been compared with the value of the body-surface obtained from the more frequently used height-weight formula. The

TABLE III.—BASAL METABOLISM AND SURFACE TEMPERATURE.

			Temperature °C.					
			Hand		Chest		Abdomen	
	Observer	No. cases	Av.	Range	Av.	Range	Av.	Range
+60% and above	E.C.W.	3	34	1·5	...	...	36·2	0·1
	E.P.P.	3	33·6	1·8	35	1·0	...	...
+40% to +60%	E.C.W.	2	32·8	0·2	33·6	0	36·2	0·5
	E.P.P.	3	32·2	11·5	35·7	1·0	...	...
+20% to +40%	E.C.W.	5	32·7	2·7	34·4	2·6	36	1·3
+10% to −10%	E.C.W.	7	31	2·4	33·1	2·9	36	2·0
	E.P.P.	5	28	11·0	32·7	3·5	...	...
−10% to −20%	E.C.W.	1	24·1	...	33·2	...	35·3	...
−20% to −30%	E.P.P.	2	21·5	1·0	34·7	1·5	...	...

maximum body-weights calculated from the stem-length, the actual body-weights, and the two surface area-calculations of the three subjects were: Ni. 164, 255 lb., 2·38, 2·19 sq. m.; K. L. 160, 252 lb., 2·02, 2·2 sq. m.; D. B. 127, 119 lb., 1·54, 1·48 sq. m. D. B., a severe diabetic under insulin treatment, was plump but not obese. There is fairly good agreement between the two methods of calculating the surface and probably no serious error was introduced into the predicted metabolism by the use of the simpler height-weight formula.

Finally, our theory of luxus consumption depends upon the respiratory exchange of fatty tissue being found to be low, but definite evidence of this is so far lacking. Lauter and Baumann [16] have argued that this is the case, because they observed that in obese patients utilization of oxygen in the upper limb was less than normal, but I cannot find that they made control observations on normal people. Dr. Dickens at my suggestion has very kindly made direct observations on fatty tissue by Warburg's method. He found that the amount of oxygen absorbed was 30 c.mm. per grm. of fatty tissue, while muscle gives a value of about 200 c.mm.—a great difference.

The hyperinsulinism theory of obesity.—There is much to be said for this theory which was, I believe, first suggested by Falta some years before insulin itself was isolated [17]. The following are reasons in its favour:—

(*a*) Obesity not uncommonly overtakes diabetic patients who are treated with insulin.

(*b*) Insulin and carbohydrate have been used for fattening lean people.

(*c*) The frequency with which diabetes occurs in obesity is compatible with the wearing out of an over-active function.

(*d*) The sugar tolerance of obesity is increased in some cases, which is compatible with an increased output of insulin; it is diminished in other cases, presumably as a preliminary to the onset of a true diabetes. In this connection I would quote the results of Beeler and Fitz [18], Allison [19]. Goldblatt, Gardiner-Hill and Forest Smith have brought forward evidence that in obesity the sugar tolerance diminishes with the age of the patient, at any rate for so-called endogenous cases. In Table IV I have tabulated from their paper the average rise of the blood-sugar above the fasting value for various classes of individuals at different ages. Undoubtedly the best measure of sugar tolerance is the area that the sugar tolerance curve makes with the horizontal, as suggested by Payne [20]. But this requires a large number of observations; instead the distance of the highest

point above the horizontal may be taken. This rise was found by Hale-White and Payne to increase on the average from 58 mg. to 67 mg. between the ages of about 30 and 70 years [21]. The endogenous cases are divided according as the obesity was present from birth, or began at puberty, or in the mother after child-birth. In the two former categories a considerable increase in the rise of 20 and 31 mg. took place after about twenty years. In the child-birth series the rise was smaller, but the results are less certain, because no note is made of the age of the mother when the obesity began.

TABLE IV.—DEXTROSE TOLERANCE TESTS.

Maximum Rise in Blood Sugar at Various Ages.

			Age	Rise mg.		Age	Rise mg.
Normal figs.	Hale-White and Payne	*Circ.*	20-40	58	...	67-78	67
Endogenous obesity ...	from birth	...	9-17	45	...	26-39	65
Goldblatt, Gardiner-Hill, Forest Smith	from puberty	...	33-39	56	...	46-59	87
	since childbirth	...	20-39	64	...	43-56	71

(*e*) It has been shown by many observers, beginning with Hamman and Hirshman (1919) [22] and ending with Lennox [23] that repeated ingestion or injection of glucose stimulates the pancreas to secrete insulin, as indicated by the fact that the blood-sugar does not rise so high after the second administration.

Dietetic treatment.—The difficulty of treating obese patients has often been noted. In five cases Strouse and Dye [24] found that the weight remained constant over a period, although the basal metabolism was greater than the calorie value of the food taken. This may perhaps be explained by the "negative" specific dynamic action which has already been mentioned.

The water content of the body may also be variable. The burning of food will make but little difference to the body weight if its place is taken up by water. The accumulation of water will be helped if much salt is taken, hence the striking results sometimes noticed after giving a salt-free diet. Grafe [25] has noted that interference with water balance is not uncommon in obese subjects, and he ascribes this to interference with the thyroid mechanism, which, according to Eppinger's view produces diuresis. In some observations by Lauter [26] the body weight volume of urine and its specific gravity, and the volume of fluid taken were noted for a long period while the patients lay in bed. A rapid fall of weight was accompanied by a rise in the volume of urine with a low specific gravity. The weight remained level or tended to rise as the volume of urine was less and the specific gravity higher. Beeler and Fitz [18] showed that if the sugar tolerance test was carried out in obese patients, and if plenty of water was given with the sugar, the volume of urine excreted during the test was less than in normal individuals. Recht [27] showed that obese patients were abnormally sensitive to pituitary injections. The volume of the urine was reduced more than in the normal, and the reduction lasted for a longer time. He also carried out the Aldrich-McClure test on patients suffering from so-called hydro-lipomatosis. In this test an intracutaneous injection of 0·2 c.c. of normal saline is made and in the normal the wheal disappears in sixty to seventy minutes, the maximum range being forty to eighty minutes. In his patients the rate of disappearance varied in different parts of the body, but it tended to be quicker, especially in the legs. This was due to the tissues being distended with fluid, which allowed rapid diffusion of the saline from the wheal. The water content of human fatty tissues varies considerably. Bozenraad [28] observed that the water content of fat was greater in wasted individuals than in fat individuals; for instance, the average water content of the

abdominal fat was 12·15% in the obese, and 31·01% in thin subjects. All these facts fit in with the view that as people become thin water becomes stored.

Anyone may be made thin with a diet of sufficiently low calorie value, especially if there are periodical fast days, but is there any special type of diet that is particularly suitable? A common practice is to allow carbohydrate and protein, but to cut the fats down severely owing to their high calorie value. But there are indications that a fat diet need not be so rigorously avoided. Gordon and Stanley [29], on the theory of hyperinsulinism, treated their patients, who were hospital nurses, with a diet of 1,200 to 1,400 calories, consisting chiefly of protein and fat given at meal times. If the patients suffered from sinking feelings and discomfort during the day, when they were taking exercise, they were given sweets made of dextrose. There are some observations by Folin [30] and by Graham and myself [31] which point to a protein and fat diet as particularly liable to cause a loss of weight in normal subjects, and many observers have noted that with such a diet obese patients do not show ketosis which would invariably result with normal people. Folin gave a diet of starch (arrowroot and cream), with or without potatoes, to normal subjects over periods varying between five to twelve days. As far as I can calculate the diet, it would appear to have contained 400 grm. of carbohydrate, 55 grm. of fat, and 7 grm. of protein, with a calorie value of about 2,180; one patient lost a kilogram, but the others remained about the same weight. I took a diet over a period of eight days containing 767 grm. of carbohydrate (including 200 grm. of lactose), and upwards of 142 grm. of fat and 8 grm. of protein in the form of arrowroot and cream. The calorie value was upwards of 4,500 (62 cal. per kg.); the weight was 0·2 kg. less at the end of the period. Graham took a similar diet of 4,686 calories (75 cal. per kg.), and the same alteration in weight was observed at the end of eight days. Hence, as is to be expected, there is no certain loss of weight with these ample carbohydrate and fat diets even though the protein content is low. We have three observations on a protein and fat diet. At the end of ten days on a diet containing 4 grm. C., 301 grm. F., 215 grm. P., with a calorie value of 3,701 (50·4 cal. per kg.), I lost 2·1 kg., and on a later occasion, after six days on a very similar diet with a calorie value of 2,347 (34 cal. per kg.) I lost 2·75 kg. After nine days on a diet containing 4 grm. C., 176 grm. F., 77 grm. P., with a calorie value of 1,969 (31·8 cal. per kg.), Graham lost 3·2 kg. in weight. Hence there does seem to be a loss of weight with a protein and fat diet even when the calorie value is enough. However, recent observations on two obese subjects have shown that the protein and fat diet has no greater effect than the mixed diet in lowering the body weight. Hence it would seem that a low calorie value is the only requisite of dietetic treatment.

Hyperthyroidism and hyperinsulinism in obesity.—From the theory of luxus consumption that Dr. Warner and I have brought forward, I have argued that hyperthyroidism is present, as well as hyperinsulinism, in obesity. But if so, it is necessary to consider why these patients do not suffer from hypoglycæmic fits due to hyperinsulinism on the one hand and from Graves' disease due to hyperthyroidism on the other. Burn and Marks [32] found that after removal of the thyroid in animals insulin hypoglycæmia was more readily produced. With thyroid feeding, on the other hand, provided the glycogen store of the liver was not diminished, the hypoglycæmic reactions due to insulin were less; in other words, the animals could stand more insulin without feeling any bad effects. Hence experimental work on physiological lines is compatible with the theory, provided we can be certain that in obesity there is ample glycogen in the liver, which is the case (see below).

The so-called "pituitary obesity" can be experimentally produced by injury to the hypothalamic region [33] and it may be due to this rather than to disturbances of pituitary function. Foster and Benninghoven [34], who produced hypothalamic obesity experimentally in animals, found that both the glycogen and the fat in the liver were increased. (This is different from diabetes, in which the fat and the carbohydrate content of the liver vary inversely, as under insulin the carbohydrate

is increased, and in untreated diabetes there is increase in the fat.) It has been shown that the production of insulin can be stimulated from the centres in the brain through branches of the vagus nerve to the pancreas, and so it is possible that in hypothalamic injury the obesity results from the increase of insulin produced reflexly in this way.

As suggested by Parkes Weber [35] among others, the exogenous type of obesity may be the result of pancreatic stimulation following the ingestion of too much carbohydrate. This may well lead to some hypertrophy of the islet tissue which will cause the blood-sugar to fall and produce feelings of hunger which must be satisfied by more food so that a vicious circle becomes established. It is one of the difficulties in treating obese patients that when the food is cut down they complain of hunger and other symptoms suggesting hypoglycæmia. As found by Davidson and Stanley, the administration of small amounts of sugar between meals relieves these symptoms, and this is a practical way of getting over the difficulty.

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Discussion.—Dr. H. GARDINER-HILL said that he must join issue with Dr. Poulton over the suggestion that hypothyroidism was not a factor in some cases of obesity, and that typical myxœdematous patients were not very fat. Obesity might result from hypothyroidism, and hypothyroid individuals, whether suffering from cretinism or myxœdema, might be either fat or thin. If he understood Dr. Poulton's argument correctly, it was based on the fact that on fasting the respiratory exchange fell but rose again as the fat intake and body weight increased, and that this rise in respiratory exchange ceased when the thyroid was removed. In spite of the failure of the respiratory exchange to continue rising after thyroidectomy, the animal or patient might still become fat; in fact it was sometimes found that animals or patients after thyroidectomy did become fat, though this was not always the case. The respiratory exchange, however, in hypothyroid patients was almost invariably subnormal.

The chief characteristic of obese patients was their excessive storage mechanism, not only for foodstuffs but for salt and water, and this might be present with an increased normal or low level of respiratory exchange. He found it difficult to believe that this mechanism depended on hyperinsulinism because during the last few years several cases of hyperinsulinism had been reported in the literature, the chief features of which were a tendency to fits, occurring in the absence of food, the presence of a low blood-sugar during the fits, and at operation or post-mortem a benign or malignant tumour of the islets of

Langerhans. As far as he could see from the reports, obesity was not a pronounced feature in these patients. Probably more light would be thrown on this excessive storage mechanism in obesity when the functions of the pars intermedia of the pituitary and perhaps the adrenal cortex were better understood. This peculiar type of storage mechanism might be inherited in cases of constitutional obesity, or acquired as a result of excessive food intake or diminished expenditure, or it might result from changes in the ductless glands. The possibility of thyroid deficient individuals being either fat or thin he had already referred to. He thought the same was true of anterior lobe pituitary deficiency. He had in mind two cases of children with suprasellar cysts destroying the anterior lobe. One of these had gained a stone or two in weight since the onset of symptoms whilst the other had become wasted. Cases of primary testicular deficiency, eunuchoidism, also might be fat or thin, though after castration there was generally a tendency for the individual to put on weight. Of thirty-one women he had seen, in whom the operation of double ovariotomy had been performed for various reasons, 84% had become fat, while of fourteen women with one ovary removed, 80% had gained in weight. In the remaining cases in each series the weight had either not changed, or they had become thin. In a number of these cases in which the basal metabolism had been investigated, it was within the normal limits. He had found the same result in eunuchoids. He thought, therefore, that some change in the storage mechanism was a more important and likely factor in the obesity of these cases than the level of the respiratory exchange.

Dr. W. W. PAYNE: Deposition of fat occurs when more food is absorbed than is metabolized. This must be brought about by an increase of foodstuffs such as sugar and fat, in the blood. There is presumably a certain threshold level or zone for these substances, above which fat is deposited and below which fat is liberated into the blood-stream in the normal; this zone as far as the blood-sugar is concerned is above the level at which hypoglycæmic symptoms occur.

Obesity would occur in any of the three following possibilities: (1) A persistently high blood-sugar or blood-fat (as insufficient is known of the blood-fat my further remarks will deal only with blood-sugar) in the group of pre-diabetic subjects and to a less extent in middle age will thus cause a deposition of fat. Insulin itself cannot be a factor in fat deposition, as in hyperinsulinism no increase of weight occurs. This is shown both in the rare cases of pancreatic tumours and in diabetics. I have observed that children who are kept for a long period with a persistently low blood-sugar do not increase in weight but do so as soon as the blood-sugar is allowed to rise.

(2) In some obese subjects it is possible that the threshold zone is lower than normal, and thus it becomes easier to deposit fat and, equally important, more difficult to liberate fat inasmuch as the lowering of the blood-sugar necessary to bring this about will cause symptoms of hypoglycæmia, in particular hunger and a feeling of weakness.

(3) It is known that in some diabetics the level of blood-sugar required to cause hypoglycæmic symptoms is higher than normal. If such a condition occurs in obese people, and work by Nissler, Tai, and Gordon [1] suggest that this is so, as soon as the body starts to withdraw from the fat deposits hypoglycæmic symptoms would manifest themselves and the subject would be driven to eat.

I suggest that one or other of these possibilities underlies many of the cases of simple obesity. The first would be easily controlled by diet, but in the second and third such treatment, owing to the unpleasant symptoms, would be too trying to the patient.

Dr. R. D. LAWRENCE that he agreed with Dr. Payne that the diabetic became obese at the onset of his disease, and often later when treated with insulin for the same reason, the blood-sugar was high during at least part of the day. Severe hyperinsulinism did not produce obesity, but he related a case in which moderate hyperinsulinism apparently associated with a vagotonic hyperactive condition of the whole intestines, produced extreme obesity of the pituitary type of distribution. The whole problem of endocrine obesity was so complicated that he could not form any hypothesis about it.

Dr. POULTON (in reply) said that if the proof of luxus consumption in obesity was sound, the hyperthyroidism was hypothetical. But he did not think that the points brought forward in the discussion were incompatible with the theory presented.

[1] Nissler N., Tai, an Shin, Gordon, B., *New York State Journal of Med.*, 1931, p. 187.

Section of Otology.

President—Mr. A. R. TWEEDIE, F.R.C.S.

[*May* 1, 1931.]

The Physical Phenomena Occurring in the Semicircular Canals during Rotatory and Thermic Stimulation.

By Prof. Dr. G. SCHMALTZ.

Offenbach am Main.

MR. PRESIDENT, ladies and gentlemen, I thank you very much for the great honour you have shown me in inviting me to give this address before your learned Society.

In seeking a subject I thought it might be interesting to you to hear something about the physical phenomena which occur in the semicircular canals under stimulation, research work with which I have been connected for some years past.[1]

If you consider the whole course of a reflex on the skeletal muscles, or the inner sensations connected with such a stimulation, it is not always very easy to say what portion is referable to the physical phenomena and what to nervous agencies in the central or other parts of the nervous system. Allow me to explain this statement by two opposite examples, one of the phenomena connected with rotation, the other with thermic stimulation. In fig. 1[2] the left-hand side represents the movement

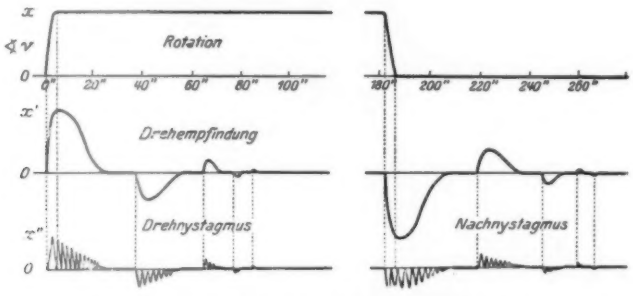

FIG. 1.—Alternating effects of rotation (Fischer, 1928).

Drehempfindung = Sensation of rotation. Drehnystagmus = Rotatory nystagmus.
Nachnystagmus = Post-rotatory nystagmus.

during rotation on a turning platform and the sensation and the nystagmus combined therewith, whilst the right-hand side shows the similar phenomena on cessation of rotation. You see represented the well-known first sensation of being turned in the same direction as the platform, and following the cessation of rotation there occurs the opposite sensation, that of being turned in the opposite direction in which the platform was turning originally.

[1] The publication of this paper has been delayed owing to the illness of the author.

[2] SCHMALTZ, Die physikalischen Grundlagen der kalorischen Vestibularreizung, *Medizinische Klinik, Wochenschrift für praktische Aerzte*, Jahrg. 1923, Nr. 15. SCHMALTZ u. VÖLGER, Ueber die Temperaturbewegung im Felsenbein bei der kalorischen Reizung des Vestibularapparates, *Pflügers Archiv f. d. ges. Physiologie des Menschen und der Tiere*, Band 204, Heft 5-6, p. 708. SCHMALTZ, Ueber die Reizvorgänge an den Endorganen des Nervus Octavus. III. Mitteilung. Die Vorgänge im Bogengang bei der kalorischen Reizung, *ibid.*, Band 208, Heft 3-4, p. 424. Id., Ueber die Reizvorgänge an den Endorganen des Nervus Octavus. IV. Mitteilung. Die Beziehung der Fischerschen Pulsionsreflexe zur Strömungsgeschwindigkeit der Endolymphe, *ibid.*, Band 217, Heft 3-4, p. 389. Id., Versuche über den Einfluss der Stellung von Kopf und Hals zum Stamm. Ueber einen von der Bauchhöhle auslösbaren Bein-Flügel-Reflex bei der Taube, *Acta Oto-Laryngologica*. Vol. XV, fasc. 2-4, p. 547.

In addition, however, an accurate observer during rotation will be able to report (Fischer,[3] Wodak, Dodge) that he also obtains temporarily the sensation of moving in the opposite direction, followed by a recurrence, though less strong, of the initial sensation. This sequence of reversed sensations may be recognizable some three times, and further alternating sensations, though reversed in character, may also be found to occur on analysis of the sensations following cessation of rotation as indicated in fig. 1 together with the nystagmus corresponding to these alternating phases. If it is asked—"Are these phenomena dependent on any physical action in the semicircular canals?" we can reply for certain that only the first phase both during and after rotation can be so controlled. In these first stages we have the endolymph movement and that of the cupula, but we may be sure that the following reversed sensations and the associated nystagmus can have nothing to do with any mechanical phenomena in the semicircular canal. For there cannot be

FIG. 2.—X-ray picture of skull of living person with lead piece put on wall of horizontal semicircular canal (Veits).

imagined any possible mechanical system consisting of a viscous liquid in a semicircular canal and an elastic highly damped body (cupula) which would perform an oscillatory movement, that would come to an entire standstill and after a certain interval start again in the opposite sense. So we feel convinced that the second and following sensation of rotation and nystagmus must be of central origin, and be connected with a central agency in the nerve itself, the ganglion or the mid-brain, though we do not know where, and of what nature it might be. This last statement will be at once accepted by all, but I now wish to demonstrate how an unexpected purely physical agency may account for the production of a phenomenon which at first sight it might be thought could only be explained biologically.

I refer to investigations made by Fischer, Wodak and Veits in Germany, on what they call "pulsion reflex" and the theoretical explanation I have given for its origin. They syringed both ears equally at the same time, and they then observed

[3] The literature concerning this question as well as the whole problems of the "regulating function" of the labyrinth will be found in a very convenient form in the book of M. H. Fischer, "Die Regulationsfunktion des menschlichen Labyrinths und die Zusammenhänge mit verwandten Funktionen," München, 1928.

that the person had a tendency to fall either forwards or backwards. This "pulsion reflex" depends on the position of the head. In the "indifferent position," where the external canal is in the true horizontal plane, one gets no reflex, even if one uses a large quantity of water in the syringing. But if you tilt the head out of that position then these "pulsion reflexes" occur. In support of these remarks I show you an X-ray picture of a head (fig. 2) in which a recent mastoid operation had been performed with a piece of lead laid on the wall of the horizontal canal. Veits [4] found in this patient that the "pulsion reflexes" ceased when he put the head in such a position that this piece of lead was horizontal. Fischer and Veits syringed during these investigations both ears with the head in the "indifferent position," and then after a time—which they varied in a series of experiments—they altered the position of the head from the "indifferent position," when they noted the occurrence of the "pulsion reflex." They observed how long the reflex lasted, taking its duration as a measurement of its intensity (fig. 3). Here you see the results of these observations both for syringing with hot or with cold water.

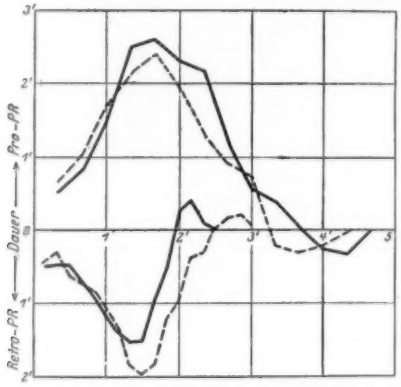

FIG. 3.—Duration of pulsion-reflexes (Fischer and Veits, 1927).

Dauer = Duration. The abscissa indicates the time between beginning of syringing and tilting the head in maximum position. The ordinates give the duration of pulsion-reflexes.

Upper curve.—Two persons each syringed with 50 c.c. H_2O of 47°.

Lower curve.—Two persons each syringed with 10 c.c. H_2O of 20°.

The abscissa is proportional to the interval between the syringing and the altering of the position of the head, whereas the ordinates show the duration of the reflex. For instance, one minute after the syringing the investigators moved the head of the person out of the "indifferent position," and they obtained a reflex which lasted about from 1 to 5 minutes. The maximum duration of the reflex took place about 1·5 minutes after syringing. After that time the reflex became slighter and slighter, till in about 3·5 minutes zero was obtained. Now Fischer and Veits observed the most astonishing fact *that after the reflex had disappeared it reappeared, but in the opposite sense;* thus there was first a *pro*pulsion reflex and then a *retro*-pulsion reflex, that is to say, the person showed a tendency to fall first forwards and then backwards. These results were a surprise to the investigators, and they sought for the correct explanation. Now two years ago I happened to have published methods based on mathematical calculations and experimental research by which it was possible to determine the speed of the endolymph fluid in the external

[4] C. Veits, Zur anatomischen Charakteristik der absoluten kalorischen Indifferenzlage, *Archiv f. Ohren-, Nasen- und Kehlkopfheilkunde*, 1928, cxviii, 305.

semicircular canal under the influence of heat or cold; and so Fischer wrote and asked me if I could determine for him the endolymph speed under the conditions of *his* experiments.

Here is the result (fig. 4). The endolymph speed is shown by the two curves on the left which I had determined according to the above-mentioned method, the first one on the supposition of a sudden (rectilinear) rise and sudden cessation of the temperature in the meatus externus, whereas the second curve ("Fall 2") represents the theoretical endolymph movement for an alteration of temperature in the meatus according to the curve which is represented on the right-hand upper corner of this chart. This curve of temperature in the meatus externus is in accordance with experimental observations under the conditions of a normal clinical test as I will describe later on. The right curve (M) on the main chart shows the intensity of

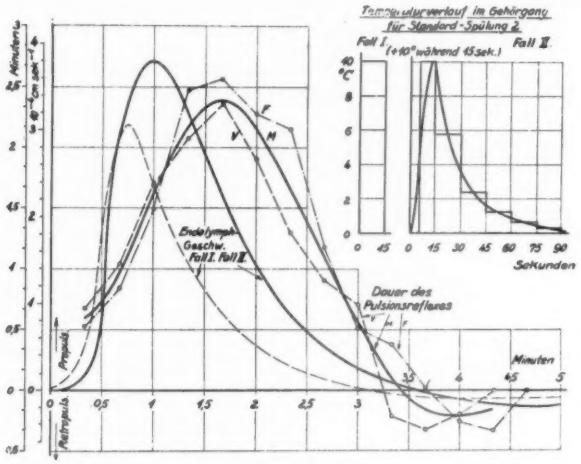

FIG. 4.—Movement of endolymph and pulsion-reflexes (Schmaltz, 1927).

Endolymphgeschwindigkeit = Endolymph speed.
Fall I = Case I.
Fall II = Case II.
Dauer des Pulsions-reflex = Duration of pulsion-reflex
Temperaturverlauf im Gehörgang = Course of temperature in the meatus externus.
Für Standard-Spülung 2 = For standard-syringing 2

the "pulsion reflex." The curves for the endolymph speed according to my calculations and the duration of the "pulsion reflex" as observed by Fischer and Veits are nearly parallel (and nearly coincide) with each other. The "pulsion reflexes" pass through the abscissa axis and become zero, and then begin to *become negative* at the end of $3\frac{1}{2}$ minutes at the same moment when the endolymph movement becomes inverse. From this it is obvious that the movement and speed of the endolymph are in conformity with the intensity of the "pulsion reflex." And thus in this case a physical basis had been found which completely accounts for a reflex phenomenon without the necessity of relying on any nebulous "biological process."

Now I will show you something of the method by which it was possible to determine the movement of the endolymph in the semicircular canal. If you

consider on a good anatomical preparation (fig. 5) the position of the postero-superior part of the meatus externus to the external semicircular canal you find that between these two points exists a bony connection ("Antrumschwelle") through which the heat can flow from the meatus externus to the canal when the former has been syringed. I have shown this supposed path of the heat by an arrow in this

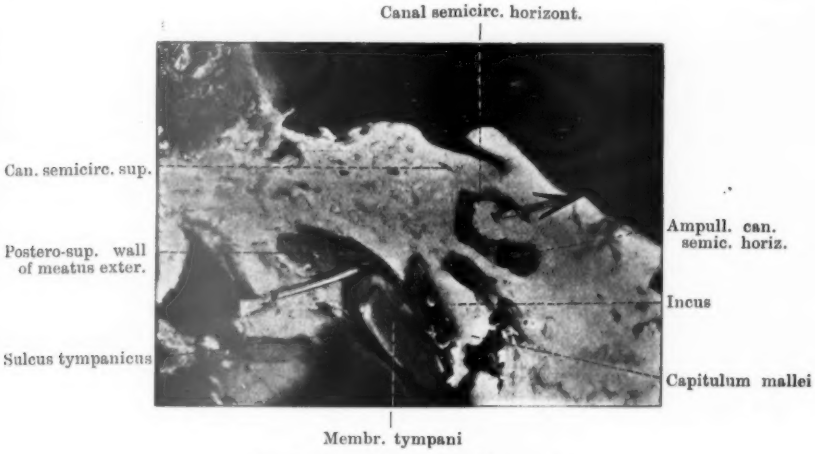

FIG. 5.—Situs preparation of inner ear.
The arrow shows the supposed path of heat from the postero-superior wall of the meatus externus to the centre of the horizontal canal.

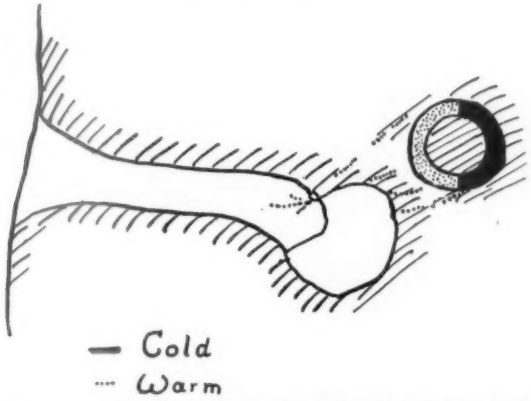

FIG. 6.—Bárány's theory of caloric stimulation. The cold liquid sinks down, the warm liquid rises.

photograph. The theory of Bárány holds, as you know, that when the meatus is heated, the wave of heat, passing in, first affects that portion of the endolymph in immediate relation with the anterior wall of the semicircular canal, whilst in the posterior portion of the canal the endolymph as it remains colder is heavier, thus a movement of the endolymph results (fig. 6) whereby the heavier part of the endolymph sinks down and the lighter one is raised.

Now in 1923, Kobrak [5] communicated the results of experiments which showed that it was possible to get all the reflexes of the thermic stimulation by a minimum degree of syringing, that is to say, only 5 c.c. of water, and differences of temperature of no more than 5°. It was doubted, however, if such small variations in temperature were really able to affect the labyrinth in the sense of the theory of Bárány. Therefore in 1924 I decided to find out by experiment whether that was true or not. I thought the only way to decide this question was to make, when syringing, delicate temperature measurements by means of thermo-couples attached on the wall of the semicircular canal. I first applied these experiments on the dead body, and then on living patients in the Clinic of Prof. Voss at Frankfurt. I constructed such an electrical thermometer with three junctions, which enabled us to measure the temperature at three different points of the body, and make comparisons of these

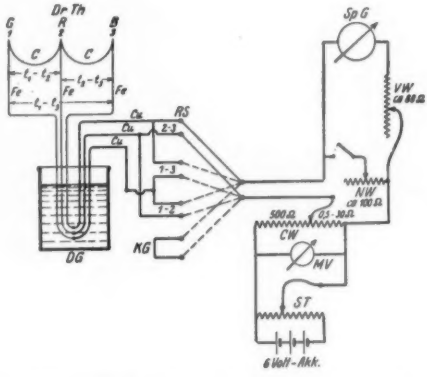

FIG. 7.—Electrical equipment for measuring the temperature in the inner ear (Schmaltz and Völger, 1924).

Dr Th = Thermocouple with 3 junctions.

Junctions:—
G1 = Meatus externus.
R2 = Rectum.
B3 = Wall of semicircular canal.
C = Constantan.
Fe = Iron.
Cu = Copper.

DG = Dewar's vessel.
RS = Switch.
KG = Shunt.
SpG = Mirror galvanometer.
MV = Millivoltmeter.
ST = Potentiometer.
NW = Shunt.

readings. Here (fig. 7) is the diagram of the electrical equipment and the arrangement of connections used in these experiments. I put the middle junction of the thermo-couple in the patient's rectum, so as to get a standard of temperature; the first of them I put in the external meatus, and the third on the wall of the horizontal canal which lay free, in the wound cavity of a recent mastoid operation. This cavity was then closed with a dressing of cotton wool, so that there should not be any change of temperature induced from the outside. By manipulating the electrical switch RS between the branches of the thermo-couple and a very sensitive mirror galvanometer I could then get measurements either of the temperature of the outer ear or the wall of the canal, or I could determine directly the difference of temperature between the semicircular canal and the external meatus. The results are shown in the following

[5] F. Kobrak, *Passow-Schäfer's Beiträge*, 1923, xix, 321; id., 1924, xx, 1; id., *Klin. Wochenschr.* 1924, iii, 195.

figures. This curve (fig. 8) shows the temperature in the meatus externus when it is heated. You see that the temperature rises very suddenly and then dies away according to a curve, which has the character of the "*e*" function. Fig. 9 shows the curve of temperature on the wall of the external semicircular canal under

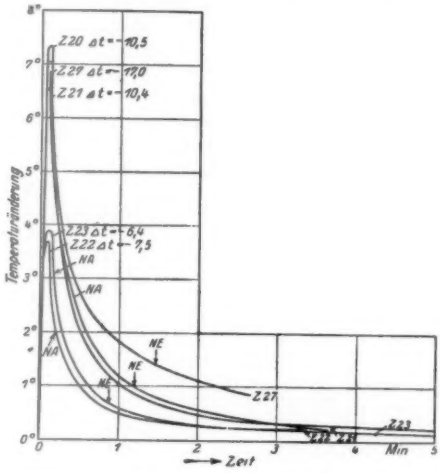

FIG. 8.—Course of temperature in the meatus externus (Schmaltz and Völger, 1924).

Temperaturänderung = Change of temperature.
NA = Beginning of nystagmus.
Δ t = Difference of temperature against temperature of body.
NE = End of nystagmus.

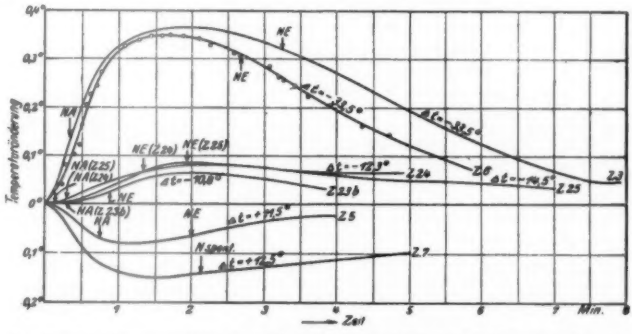

FIG. 9.—Course of temperature on wall of the horizontal semicircular canal on a living person (Schmaltz and Völger, 1924).

Temperaturänderung = Change of temperature in temporal bone on the wall of horizontal canal
Zeit = time.
Δ t = Difference of temperature of water with which syringing took place against temperature of body.
NA = Beginning of nystagmus.
NE = End of nystagmus.

different conditions, according to a long series of experiments. The upper curves show the change when "cold" is applied and the lower ones show the changes following heating. It will be seen that the points of the different single observations which are indicated thus *o* are situated very closely to the equalized curve. It

appears from these diagrams that a difference of temperature of 33° against the temperature of the body applied to the meatus externus corresponds with a maximum drop of the temperature of the canal of only 0·35°. The beginning and end of the nystagmus is indicated in the figure by NA and NE. The nystagmus sets in regularly after about 0·2 to 0·3 minutes, but the nystagmus stands, generally, on a higher level of temperature than it started. Perhaps some nervous fatigue phenomena would account for this fact.

Fig. 10 shows the changes in the temperature in the internal canal with "mass syringing," that is to say, syringing with large quantities of water, such as 1,000 c.c. of about 10°. The temperature drops about 4°, then rises gradually and regularly. The lower curve corresponds with an experiment in which we first syringed with large quantities of water, and then with minimal syringings at 9, 12, 18, 22, 29 minutes. It will be noted that every new appearance of nystagmus is connected with a definite wave of temperature on the original curve. Then I made direct measurements of the differences of temperature between the meatus externus and

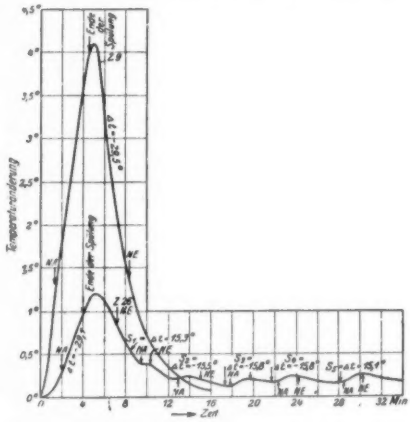

FIG. 10.—Course of temperature in the temporal bone. Experiments with living persons, mass-syringing (Schmaltz and Völger, 1924).

Temperaturänderung = Change of temperature on wall of semicircular canal.
Zeit = Time.
NA = Beginning of nystagmus.
NE = End of nystagmus.
Ende der Spülung = End of syringing.
The syringing took place with 1,000 c.c. H_2O of 29° below temperature of body.

the wall of the external semicircular canal. For these measurements the connections were arranged so that the electromotive forces of the two thermocouples G and B are in opposite directions. So they partly compensate each other and the galvanometer reading gives directly the difference of temperature between the two junctions B and G, i.e., in our case the meatus externus and the semicircular canal.

The results of these experiments are represented in fig. 11. The difference of temperature between the meatus externus and the wall of the semicircular canal first rises, then falls. The experiment showed that in 3½ minutes this difference of temperature becomes negative, that is to say, after this period the semicircular canal is warmer than the meatus externus. This accounts for the change of flow of heat already mentioned, so that the heat flows first inwards and then outwards. That furthermore is the reason for the change of direction of the movements of the endolymph, and evidently also for the change from propulsion reflex to retropulsion in the observations by Fischer and Veits.

Now I wish to show you very briefly how it is possible to find theoretically, i.e., by numerical and graphical methods, the speed and the displacement of the endolymph in the semicircular canal when the change of the temperature in the meatus externus is given. The first task we have to fulfil for this purpose is to determine the change of the temperature in the centre of the canal under given external conditions. Then later on we will see how it is possible to find from this the speed and the displacement of the endolymph connected herewith.

The flow of heat in one direction of a homogeneous body (that the labyrinth bone is not exactly homogeneous is one of the deviations of actual conditions from the theoretical suppositions which we cannot account for) is ruled by the well-known differential equation:

$$\frac{d\,\theta}{d\,t} = a\,\frac{d^2\,\theta}{d\,x^2}$$

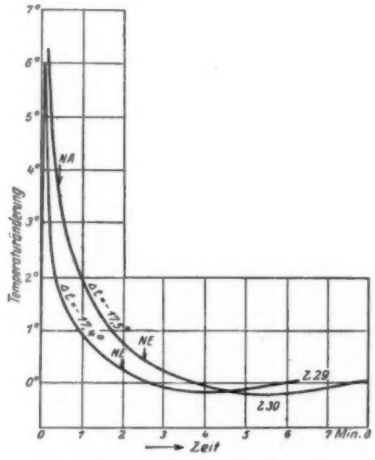

FIG. 11.—Difference of temperature between meatus externus and semicircular canal (Schmaltz and Völger, 1924).

Δt = Difference of temperature of water against temperature of body.
Temperaturänderung = Difference of temperature between meatus externus and semicircular canal.

where "θ" is the temperature and "a" the thermal conductivity of the body. One integral of this equation is

$$\theta_x = \theta_c\,\frac{2}{\sqrt{\pi}}\int_0^{\frac{x}{\sqrt{4\,a\,t}}} e^{-\frac{x}{4\,a\,t}}\,d\left(\frac{x}{\sqrt{4\,a\,t}}\right) = \theta_c\;\Phi\,(x_1\;t)$$

This integral applies to the case, that the temperature at the end of a one-dimensional continuous body drops suddenly from the value θ_c to zero. This gives the temperature θ_x as a function of the two variables x (distance from the beginning from the one-dimensional body) and the time t.

The numerical values of this integral can be found in tables and easily dealt with. They give us the curve of temperature as a function of time for any given distance from the source of heat, i.e., in our case the spot in the meatus externus where the syringing takes place. It is no use to insist upon the theoretical details. I will show you only the results for conditions which correspond with the actual value of the conductivity of heat of the temporal bone as I have determined it experimentally in my laboratory.

Fig. 12, Plate I, shows the temperature field which occurs in a temporal bone when at the crossing-point of the three co-ordinate axes the temperature is raised suddenly. The axis from the left to the right corresponds to the time, whereas the axis from backward to forward corresponds to the distance from the source of heat. You see that the curve of temperature in relation to the time which is in the plane of the source of heat, as we supposed, rectilinear, becomes flatter and flatter the farther away we go from the source of heat and changes its character nearly completely. By superposing, i.e., adding graphically small steps of rising or dropping temperature, it is possible to find the curve of temperature in the bone for any given distance from the source of heat and for any given character of the initial change of temperature in the plane of the source of heat. Fig. 13, Plate I, gives an idea of this method. Fig. 14, Plate II, shows the change of temperature in two planes of different distance from the source of heat when there the temperature rises and then drops suddenly. If you consider the course of temperature in *this* plane which has a distance of 5 units from the source of heat, you have the red curve (θx_1); and if you go further away, on this blue plane, you get a temperature curve (θx_2) which is flatter. After a certain time these two curves attain the same point, that is to say, there is, at this time, the same temperature in the red plane and in the blue plane. And afterwards the temperature of the blue plane, which is further away, becomes higher than in the red plane. This accounts for the change of flow of heat from the forward-backward to the backward-forward direction.

After having given an outline of a method by which to find the curves of temperature in any desired plane of the labyrinth bone for any given curve of temperature in the meatus externus I propose to show briefly how it is possible to find from this the actual endolymph speed in the semicircular canal.

Fig. 15 shows the theoretical deductions necessary to solve the problem. You see that the result is very simple. If we know the "gradient" of temperature, that is to say, the value $\frac{d\theta}{dx}$ for the centre of the semicircular canal for any given moment (and our described method above enables us to do so by differentiating the integral $\theta_x = \Phi(x, t)$ after x) the product of this quantity by the coefficient ξ gives us the velocity of the endolymph. The coefficient ξ contains the following quantities: the density of the endolymph fluid, its coefficient of thermal dilatation, its coefficient of viscosity and the geometrical measurements of the semicircular canal.

Figs. 16 and 17 show the results of the adaptation of this theoretical method to the experimental determination of the temperature in the inner and outer ear as I have shown previously. On the first diagram the upper curve shows the temperature on the wall of the semicircular canal after syringing according to three of my experiments. The second line below shows the speed of the endolymph connected herewith as it was found by the method described above. The third line below shows the displacement of the endolymph. This curve is evidently the integral curve of the curve in the middle of our chart giving the speed as function of the time. The speed of the endolymph has its maximum after $1\frac{1}{2}$ minutes, and attains the small quantity of $20 \cdot 10^{-6}$ cm./sec. The total displacement of the endolymph after four minutes when the speed has become nearly zero is about $35 \cdot 10^{-4}$ cm. ($35\,\mu$). The curves correspond to changes of the temperature in the meatus externus of about 12° against the temperature of the body.

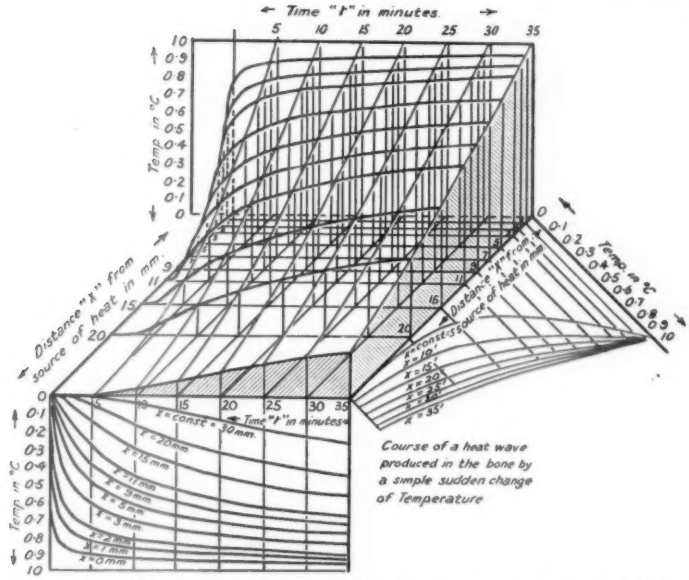
Time "t" in minutes
Temp in °C
Distance "x" from source of heat in mm
Course of a heat wave produced in the bone by a simple sudden change of Temperature

FIG. 12.—Temperature field in continuous body, giving temperature as function of distance from source of heat and time (see p. 10).

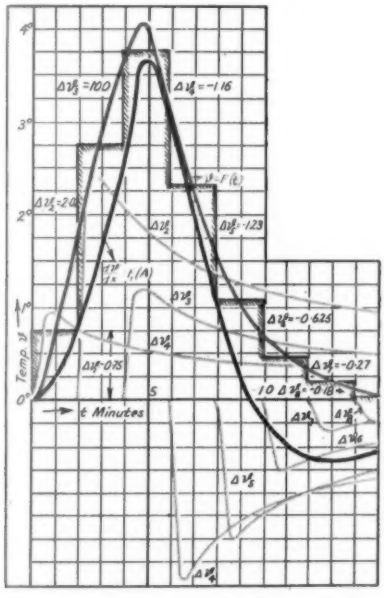

FIG. 13.—Determination of temperature in the inner ear. Red curve: $\theta = F(t)$: change of temperature in the meatus externus. Green curve: rectangular steps of raising and falling temperature replacing continuous red curve. Yellow curve: values of $\frac{d\theta_x}{d_x}$ for each step of green line. Blue curve: sum. = superposition of yellow curves (see p. 10).

PLATE II.

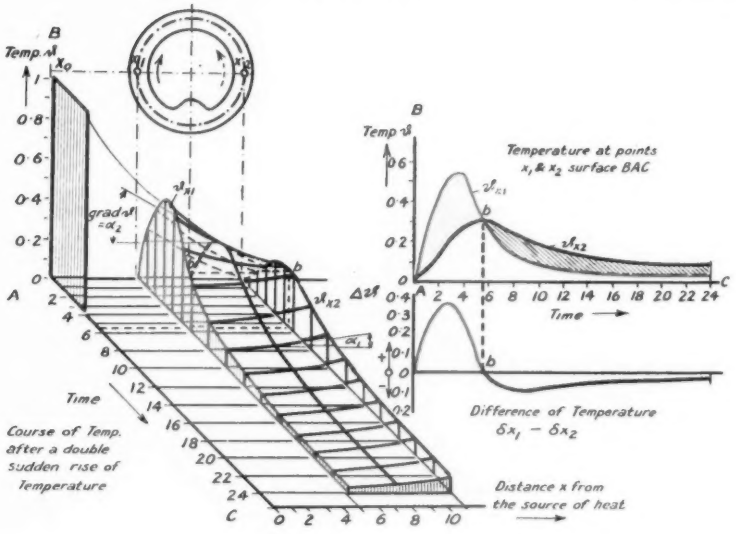
Temperature at points x, & x2 surface BAC
Time
Difference of Temperature
Course of Temp. after a double sudden rise of Temperature
Distance x from the source of heat

FIG. 14 (see p. 10.)

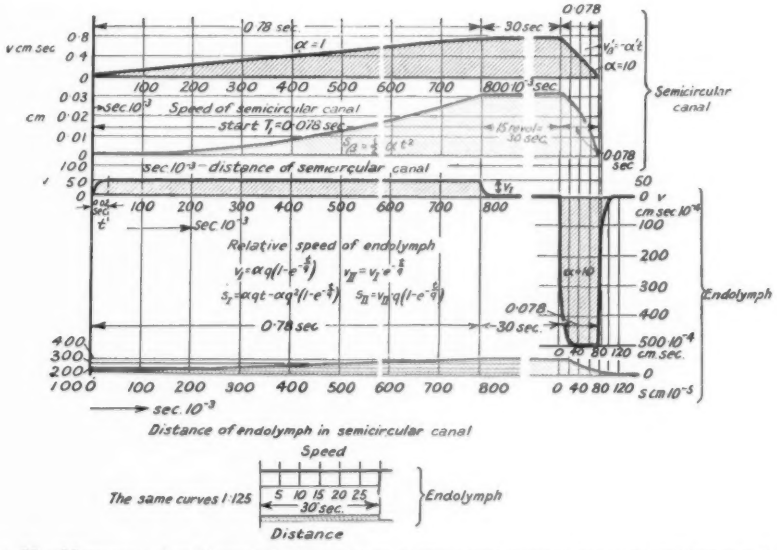

FIG. 20.—Movement of endolymph during rotation of semicircular canal (see p. 14 and footnote 8).

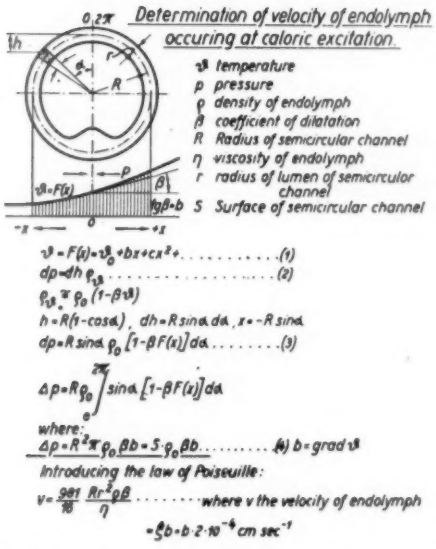
Determination of velocity of endolymph occuring at caloric excitation
ϑ temperature
p pressure
ϱ density of endolymph
β coefficient of dilatation
R Radius of semicircular channel
η viscosity of endolymph
r radius of lumen of semicirculor channel
S Surface of semicircular channel
ϑ = F(x) = ϑ0 + bx + cx² + ... (1)
dp = dh ϱ0β ..... (2)
ϱ0β = ϱ0 (1 - βϑ)
h = R(1 - cosα), dh = R sinα dα, x = -R sinα
dp = R sinα ϱ0 [1 - βF(x)] dα ..... (3)
Δp = Rϱ0 ∫ sinα [1 - βF(x)] dα
where:
Δp = R²π ϱ0 βb = S ϱ0 βb ..... (4) b = grad ϑ
Introducing the law of Poiseuille:
v = 981/16 · Rr²ϱβ/η ..... where v the velocity of endolymph
= ϱb · b · 2 · 10⁻⁴ cm sec⁻¹

FIG. 15.—Determination of velocity of endolymph occurring at caloric excitation (Schmaltz, 1931).

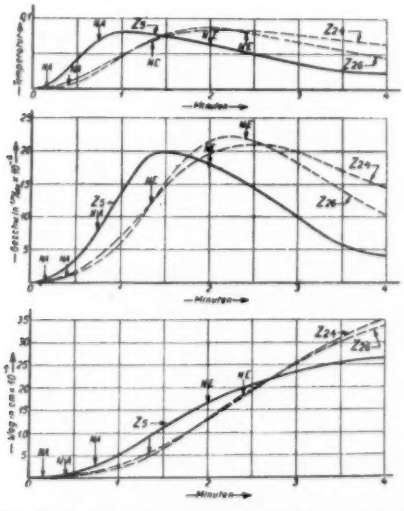

FIG. 16.—Movement of endolymph occurring at minimum (5 c.c.) syringing (Schmaltz, 1925).

Gesch. = Velocity.
Weg = D splacement.
NA = Beginning of nystagmus.
NE = End of nystagmus.

Fig. 17 shows the same data for the so-called mass syringing. So much about caloric stimulation.

Now I will tell you something of the theory of the rotatory test. You know the classic conception of Mach, which I think still holds good. He says that when you rotate the canal, either around its centre, or (which is, as far as the movement of the endolymph in a *rigid* canal is concerned, the same) around an eccentric axis, then during the period of acceleration the endolymph remains by force of inertia behind the movement; and when the movement stops the endolymph goes forward again. You can see this movement on this model which I turn during the starting and stopping [demonstration].[6] And there can be observed

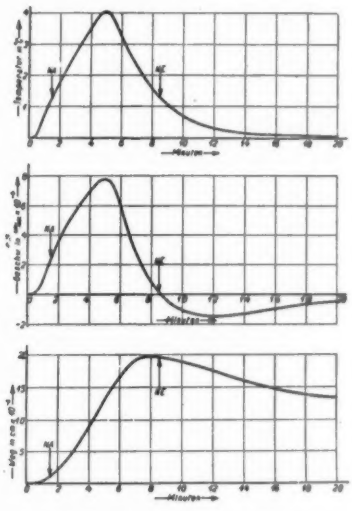

FIG. 17.—Movement of endolymph occurring at mass-syringing (1,000 c.c. H_2O). Difference of temperature against body minus 29° (Schmaltz, 1925).

Geschw. = Velocity. Weg = Displacement.

the interesting fact that you get the same movement of the wheel in comparison to the plane which holds the axis whether you turn on a small or on a big radius. This is a fact which took much room in the older literature, and was often doubted by authors not acquainted with the well-known facts of kinematics. The centrifugal force has no effect on the movement of the endolymph as long as we consider the walls of the membranous canal as rigid. For exact work, of course, we are not allowed to make this supposition. As a matter of fact Lorente de Nó has found certain features of the reflexes upon rotation which can only be understood as the effect of centrifugal forces upon the liquid contents of the labyrinth.[7]

6 The author demonstrated to the audience a model consisting of a symmetrically built wheel of about 3 in. in diameter, mounted on a very fine axis, and to which was attached a very light indicator.

7 *Ergebnisse der Physiol.*, 1931, xxxii, Lorente de Nó, "Ausgewählte Kapitel aus der vergleichenden Physiologie des Labyrinths," see cap. 6: "Die Reflexe infolge der Wirkung der Zentrifugalkraft"; cap. 7: "Ueber die dritte Drehreaktion."

Fig. 18 gives a short outline of the theory of endolymph movement when the semicircular canal is rotated. The main idea is this: In the case of a body being accelerated, its mass, multiplied by the acceleration, equals the outer force which is acting on the body, in our case the force of the friction. This latter equals the coefficient of friction, multiplied by the speed itself. This is the fundamental equation (1) by which this figuring is done. With integration of this differential equation very simple results are given. During acceleration the relative speed of the endolymph is proportional to the acceleration of the canal and the quantity q This quantity contains, as the equation (4) shows, the dimensions of the canal the density and the viscosity of the endolymph. The speed of the endolymph rises very quickly to its maximum. The ratio of this rising of the endolymph speed depends according to equation (1) only from the term $\left(1-e^{-\frac{t}{q}}\right)$ and therefore is *independent of the acceleration* of the canal.

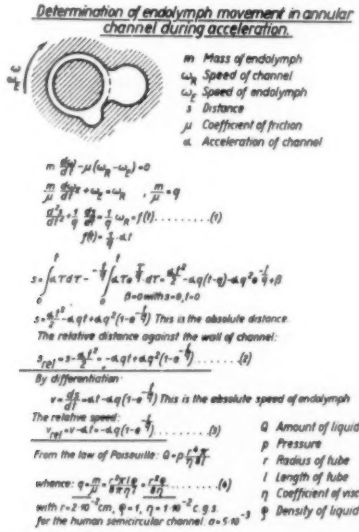
Determination of endolymph movement in annular channel during acceleration.
m Mass of endolymph
ω_R Speed of channel
ω_E Speed of endolymph
s Distance
μ Coefficient of friction
α Acceleration of channel
Q Amount of liquid
p Pressure
r Radius of tube
l Length of tube
η Coefficient of viscosity
ϱ Density of liquid

FIG. 18.—Determination of endolymph movement in annular canal during acceleration (Schmaltz, 1931).

Fig. 19 shows this phenomenon more clearly. The relative speed of the endolymph rises according to the term $\left(1-e^{-\frac{t}{q}}\right)$ very quickly and attains its maximum already in about 0·02 sec. Then it becomes constant until the acceleration of the canal has come to its end, that is to say until the speed of the canal has become constant. Then the speed of the endolymph against the canal dies away according to the same law by which it had risen. It is zero about one-fiftieth of a second after the acceleration has come to an end. Then the endolymph remains unmoved relative to the canal and rotates with the same absolute speed as the canal itself until the canal stops. Then during the deceleration period the movement of the endolymph in the canal takes place in the opposite sense. The displacement of the endolymph (curve S) becomes straight after one-fiftieth of a second, then it rises until the acceleration period is finished and the speed has died away, and then the displacement becomes constant, that is to say, the endolymph when it has been displaced in the canal stops in its new position. During

the deceleration (stopping) period the liquid is displaced in just the opposite way independently from the time during which the movement comes to an end. The result is that however the canal may be started or stopped the liquid contained in it is found at the same place as before when the movement has come to an end.

Fig. 20 (plate II) gives an idea of the movement of the canal and the endolymph under the conditions of an ordinary clinical rotation test. In drawing this diagram it was supposed that the movement of the platform was started very slowly within an angle of about 70°, that the rotation was then continued with constant speed during 30 secs., and that the platform was then stopped very suddenly within an angle of about 7°. The first curve from above shows the speed of the platform, the second the displacement of same,[8] the third shows the relative speed of the endolymph and the fourth the displacement of the endolymph in the canal.

The speed of the endolymph under the above-mentioned conditions attains the value of $500 \cdot 10^{-4}$ cm./secs.

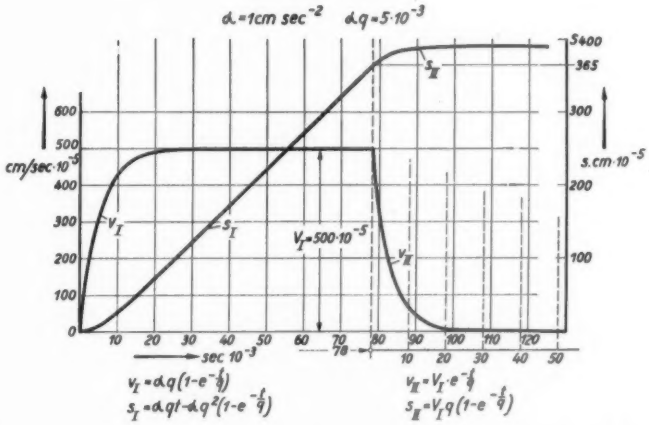

FIG. 19.—Endolymph movement during rotatory acceleration of semicircular canal (Schmaltz, 1931).
V_I and V_{II} = Speed of endolymph. S_I and S_{II} = Displacement of endolymph.
The abscissæ show the time in sec. 10^{-3}.
The ordinates show the speed in cm./sec. 10^{-5}, and the displacement in cm. 10^{-5}.

The two methods for the determination of the endolymph movement during the ordinary rotatory test, conditions which I have described before, and the caloric test (10 c.c. water of about 10°), and it is interesting to compare these two results (fig. 21). The endolymph speed is lower in the caloric test than in the rotatory test, but it lasts much longer. It is astonishing that these two kinds of stimulation give reflexes so nearly alike, and that shows on what a broad scale the labyrinth reacts to stimulations. The same applies to the eye, as you know.

It is very difficult to say what happens in the ampulla during the movement of the endolymph. Up to the present it has been considered that the cupula had

[8] In preparing this figure an error of the draughtsman has, unfortunately, been overlooked by the author. The 2nd curve gives the displacement of the canal during the period of constant rotation (30 secs.) as constant, whereas evidently this displacement increases linearly from the point 800·10-3 secs. up to the point 800·10-3 secs. + 30 secs. (end of the constant rotation) and then drops according to the parabolic law. The other curves are correct.

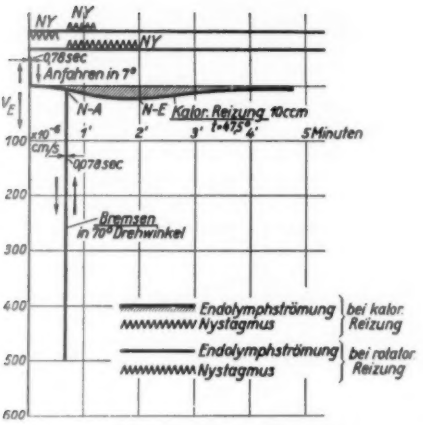

FIG. 21.—Comparison between endolymph current occurring at rotatory and caloric excitation (Schmaltz, 1931).

Rotary excitation : angular speed 180°/sec.
Onset : in 0·78 sec. = 70°.
Stopping : in 0·078 sec. = 7°.
Caloric excitation : 10 c.c. H_2O t = 47·5°.
Endolymphströmung = Endolymph current.
bei kalorischer Reizung = Occurring at caloric excitation.
bei rotatorischer Reizung = Occurring at rotatory excitation.

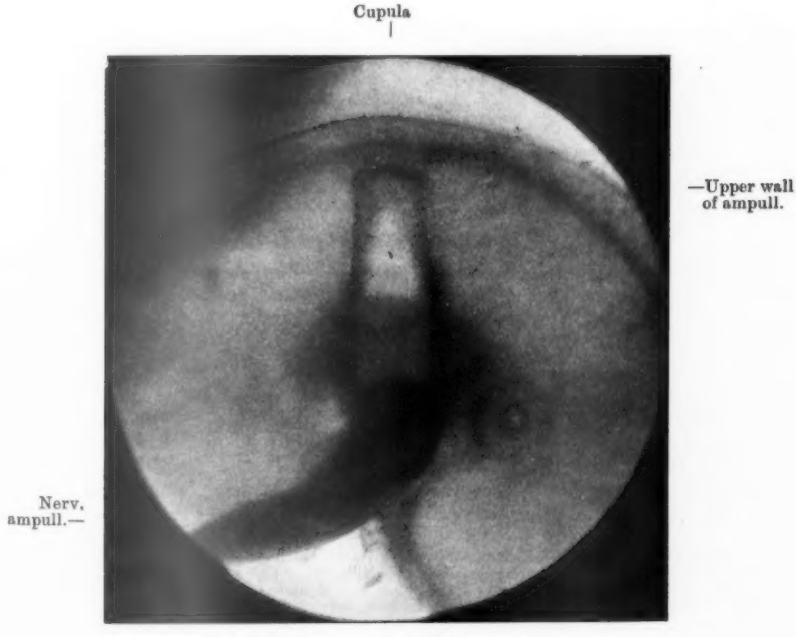

FIG. 22.—Cupula of pike (Steinhausen, 1931).

a rather strong directional force, that means a determined frequency of oscillation, but that is not so. Steinhausen, in Greifswald, was able to prepare the ampulla of a pike so that it was possible to observe the cupula a very short time after the death of the animal, and to see what happens with it on rotation or even caloric stimulation.[9] Steinhausen has shown to me in his laboratory under the microscope that when the canal is rotated, a very quick deviation of the cupula occurs, which is of the character of the movement of the endolymph as shown in the equation I have given. On heating the Ringer solution in which the preparation of the fish was contained at a definite spot by means of a thermocauter, Steinhausen got deviations of the cupula which lasted some minutes, and then very slowly disappeared. I am glad to find that Steinhausen's work corresponds with mine as regards the caloric and rotatory tests. I show you here a photograph of a preparation of a pike's cupula made by Steinhausen (fig. 22).

Speed of endolymph and rate of stimulation occuring at different tests of labyrinth.

Author	Kind of stimulation	Observation	Maximum Velocity of Endolymph v_E cm sec.	Duration of stimulation T sec.	Rate I of stimulation $v \cdot T$ cm	Rate II of stimulation $v \cdot \sqrt{T}$ cm
		Rotatory Tests.				
Dohlmann	Turning chair	Minimum deviations of eyes	$21{\cdot}5 \cdot 10^{-6}$	0,2	$4{,}3 \cdot 10^{-6}$	$9{,}7 \cdot 10^{-6}$
Mulder	Turning chair	threshold of sensation of rotation	$45 \cdot 10^{-6}$	0,8	$36 \cdot 10^{-6}$	$12{,}7 \cdot 10^{-6}$
Dodge	Turning chair (accelerated rotation)	Sensation of rotation in 65% of experiments	$48{\cdot}5 \cdot 10^{-6}$	13,3	$64{\cdot}5 \cdot 10^{-6}$	$17{,}2 \cdot 10^{-6}$
	Harmonic oscillation of 10°	Sensation of oscillation in 85% of experiments	$7{\cdot}5 \cdot 10^{-6}$	8	$60 \cdot 10^{-6}$	$21{\cdot}2 \cdot 10^{-6}$
Van Rossem	Turning chair	Threshold of sensation	$1790 \cdot 10^{-6}$	0,022	$39{\cdot}4 \cdot 10^{-6}$	270
Buys	Turning chair	Threshold of nystagmus	$43 \cdot 10^{-6}$	?		–
Schmaltz (estimation)	Normal turning of head when observing lateral object 70° in 1 sec. stop: 2°		$3400 \cdot 10^{-6}$	0,05	$172 \cdot 10^{-6}$	$778 \cdot 10^{-6}$
Normal clinical rotatory test	Turning chair 180°/sec. - stoping in 7°	Strong nytagmus	$50\,000 \cdot 10^{-6}$	0,078	$3900 \cdot 10^{-6}$	$14\,000 \cdot 10^{-6}$
Buys	Sudden wheeling round of head 65° in 0,25 sec.		$176\,000 \cdot 10^{-6}$	0,05	$8800 \cdot 10^{-6}$	$39\,400 \cdot 10^{-6}$
		Caloric Tests.				
Fischer u. Veits Schmaltz	Minimum caloric test on both ears	Threshold of „pulsion-reflex"	$15 \cdot 10^{-6}$	2,5	$37{,}5 \cdot 10^{-6}$	$23{,}8 \cdot 10^{-6}$
Schmaltz	Caloric tests	Threshold of visually observed nystagmus	$5{\cdot}5 \cdot 10^{-6}$	22	$121 \cdot 10^{-6}$	$25{,}8 \cdot 10^{-6}$
Schmaltz	Caloric test with large quantities of water	Threshold of visually observed nystagmus	$135 \cdot 10^{-6}$	~ 60	$8\,100 \cdot 10^{-6}$	$1040 \cdot 10^{-6}$

Schmaltz 1931	Stimulation of Labyrinth	S.La 35a

FIG. 23.—Stimulation of labyrinth under different conditions.

Fig. 23 may be of interest as giving a comparative record of the speed of the endolymph in connection with the different experiments and observations on the rotatory test as described in literature, and based on the equation already given ruling this speed.[10] One sees from this table that not only the speed of the endolymph varies to a great extent, but that under the conditions under which the different investigators have worked, the time (T) during which the flow of the endolymph was stimulating the end-organ was also varying within wide limits.

9 W. Steinhausen, "Zur Histologie und Physiologie der Cupula terminalis in den Bogengangsampullen des Labyrinths," *Folia oto-laryngologica*, 1929, xvii, 410; id., "Ueber den Nachweis der Bewegung der Cupula, etc.," *Pflüger's Archiv f.d. ges Physiologie des Menschen und der Tiere*, 1931, ccxxviii, 322; id., *Zeitschr. f. Hals-, Nasen- u. Ohrenheilkunde*, 1931, xxix, 212.

10 The literature used in computing this table will be found together in the book of Fischer, loc. cit., and in the publications of the author given in footnote (2).

Let us take, for instance, a normal clinical test, in which the turning platform is turned with the speed of 180°/sec. and then is stopped within an angle of 7°. In this case the endolymph speed will attain a maximal value of $50000 \cdot 10^{-6}$ cm./sec. The time during which the flow of the endolymph is acting will be in this case only 0·078 sec. In another case which took place in the work of Dodge, who experimented with a very slowly turning chair, we find endolymph speeds of only $48 \cdot 5 \cdot 10^{-6}$ cm./sec., but which acted during 13·3 secs. The question arises, which will be the appropriate expression for the amount of stimulation of the end-organ considering the speed of the endolymph and the time during which it is acting. The simplest hypothesis we might make would be, that slow speed acting during long time would have the same effect as high speed acting during a short time. This would mean that the term V.T would be a correct rate of stimulation. But I think we have some reason to believe that this expression does not indicate the facts correctly enough and that the influence of high speed of endolymph acting during a short time upon the end-organ is higher than the influence of a high speed acting a short time. Therefore it might perhaps fit the facts better if we form the term $V.\sqrt{T}$ as the rate of stimulation. This term would be similar to certain well-known laws of the electric stimulation of the nerve. I have put both terms into this table to give an idea of the differences of stimulation under different conditions. If you look through the table you find first the experiments of Dohlman in Sweden. Dohlman recorded with a photographic method the smallest deviations of the eyes which could not be observed directly. In the next two lines you find the results of the experiments of Mulder and Dodge. Mulder, as well as Dodge, did not observe the deviations of the eyes but the appearance of the smallest perceptible sensations of rotation. It is very interesting to see that the stimulation which is necessary to create such sensations is somewhat higher than the stimulation necessary to create the smallest deviations of the eyes observed by Dohlman. Further down you find the normal clinical test as described before, and you see that this stimulation is about 1,000 times higher than the stimulation which creates the first sensation of rotation. Then come the results of the experiments of Buys, who studied the rotatory acceleration when making voluntary movements with the head, for instance when looking suddenly from one side to the other. It is very interesting to see that such movements cause stimulations, which are about two or three times higher than those in the normal clinical tests. Then are shown in our table the stimulations by the caloric test, which, as I have stated before, now for the first time can be compared with the stimulation by the rotatory test. You see that the finest reflexes, which have been produced by the caloric test (the "pulsion-reflexes" by Fischer and Veits) are connected with stimulations of about the same quantity, as the smallest rotatory stimulations of Dohlman and Mulder. The stimulations connected with an ordinary clinical test, as I have studied in my investigations of the temperature changes in the inner ear, have about the same quantity as the stimulation in the test of van Rossem.

In the next table (fig. 24) I give the endolymph speed and its duration connected with rotation under different conditions. The first column of the table shows the time (Z) necessary to produce one rotation on the rotating platform. You see that this time in our table varies from 360 sec., which means a very, very slow rotation, to 0·6 sec. which means a very quick rotation. The next column gives the angular speed (ω_o) connected herewith in °/sec. In the headings of the following columns you find the angles in which our supposed turning table might be stopped. So for instance if you take the third column in the eighth line,[11] you have the case when a turning table rotating with the ordinary speed of one revolution in 2 sec. might be stopped within an angle of $\beta = 5°$; and then you find from our table that in such a case the endolymph speed according to the equation I have shown before attains the value of $70 \cdot 10^{-3}$ cm./sec., and that this endolymph speed is working upon the

[11] Second field of the table.

cupula during the time T = 0·055 sec. So it is possible to find in this table, for any given case of speed of the rotatory platform, and of slower or quicker stopping or starting of it, the endolymph speed and the time of stimulation connected with such an experiment. Now I have marked upon this table also the different experimental verifications of this ideal experiment. The area labelled "A" corresponds with the experiments of Dohlman, giving the threshold for the deviations of the eyes. The next area, "C" corresponds with the threshold for the sensation of rotation as observed by Mulder, Dodge, and van Rossem. The area marked "D" corresponds with the conditions of the ordinary clinical rotatory test. Now, as I have described before, it is possible to figure out and determine the speed of the endolymph, and its duration under given conditions on the caloric test. Therefore, it is possible to compare these caloric tests with the rotatory, that is to say, we can tell now, what experimental conditions upon the rotatory chair we must realize in order to get approximately the same conditions as under caloric stimulation. So the area marked "E" shows the endolymph movement connected with the pulsion-reflexes of Fischer and Veits, and the area marked "F" the small caloric tests as investigated by myself. The white areas in this table are unexplored. I have also figured out approximately what stimulation of the end-organ in the semicircular canals might be expected under different conditions of ordinary life. So for instance in the area No. 1 you see the endolymph speed connected with the rolling of a ship in good weather. The table gives you now an indication of the conditions which must be employed to obtain with the turning chair approximately the same stimulation of the semicircular canals as you would have in a rolling ship. No. 5 gives another example, i.e., the stimulation of the semicircular canals when you are in a railway train which goes suddenly in a curve. No. 7 gives the stimulation which attacks the inner ear when you suddenly wheel round your head in looking from one side to the other. The most interesting result of this table is, that it gives an idea of the different movements of the endolymph connected with rotatory test and caloric test in different conditions in ordinary life.

Discussion.—Dr. DAN MCKENZIE said that with regard to the problems associated with the semicircular canals he found great difficulty in explaining all the rotation and caloric phenomena on physical grounds. His chief difficulty lay in the fact that the calibre of the semicircular canal was so minute. Earlier theorists talked of endolymph "flowing" round the canal with a lagging behind and a stoppage of the movement later on. They said that after the rotation had lasted a considerable time the endolymph in the canals became, as it were, "frozen together with the walls," and then the nystagmus stopped. That theory assumed the existence of a completely circular tube, round which the endolymph could circulate. But the vestibule formed part of the circle, and it was a comparatively large cavity, into which the fluid flowed, so that there could be even circulation round the tube. Physical explanations had to meet that fact. Therefore, one should not speak so much about the "circulation" of the fluid as about variations in its pressure in the ampulla. The end-organ of the semicircular canals was extraordinarily sensitive, and there were doubtless changes of pressure which would be incapable of physical proof. Hence a neurological aspect came into the question, the stimulation of a hyper-sensitive structure and its subsequent exhaustion. The question was thus more complicated than would appear on a mere physical view. There were three canals in the temporal bone, and in certain circumstances they were opposed to each other. One might be stimulated without the others sharing in it except in a trifling manner, so there was an interplay of hyper-stimulation and exhaustion. He had worked out the effects of stimulation and of exhaustion of the semicircular canal ampullary nerve-organ, and it was as easy to explain it from that point of view as physically. But in these cases it was, when all was said and done, difficult to be sure of the facts. In examining people, it was difficult to know definitely when nystagmus began and when it stopped. He had the feeling that in dealing with living people and tissues and functioning organs, all manner of subsidiary and secondary differences and interferences interposed themselves and so led to insecurity when attempting to find a basis for theories.

VARIOUS STIMULATIONS OF SEMICIRCULAR CANALS.

Z sec.	ω_o grad.	$\beta = 0{,}5°$ $aq = v$ cm./sec.	T sec.		vT cm.	$v\cdot\sqrt{T}$	$\beta = 1°$ $aq = v$ cm./sec.	T sec.		vT cm.	$v\cdot\sqrt{T}$
360	1	$21{,}5\cdot10^{-6}$	1,0	A	$21{,}5\cdot10^{-6}$	$21{,}5\cdot10^{-6}$	$10{,}8\cdot10^{-6}$	2,0	A	$21{,}5\cdot10^{-6}$	$15{,}27\cdot10^{-6}$
180	2	$86\cdot10^{-6}$	0,5	C	$43\cdot10^{-6}$	$61\cdot10^{-6}$	$43\cdot10^{-6}$	1,0	C	$43\cdot10^{-6}$	$43\cdot10^{-6}$
120	3	$194\cdot10^{-6}$	0,33	**6**	$64\cdot10^{-6}$	$111\cdot10^{-6}$	$97\cdot10^{-6}$	0,66		$64\cdot10^{-6}$	$79\cdot10^{-6}$
72	5	$337\cdot10^{-6}$	0,20		$107\cdot10^{-6}$	$151\cdot10^{-6}$	$268\cdot10^{-6}$	0,4		$107\cdot10^{-6}$	$169\cdot10^{-6}$
60	10	$21{,}5\cdot10^{-4}$	0,1		$2{,}15\cdot10^{-4}$	$68\cdot10^{-5}$	$10{,}8\cdot10^{-4}$	0,2		$2{,}15\cdot10^{-4}$	$48{,}3\cdot10^{-5}$
12	30	$19{,}4\cdot10^{-3}$	$33\cdot10^{-3}$	**3**	$64\cdot10^{-5}$	$3{,}52\cdot10^{-3}$	$9{,}7\cdot10^{-3}$	$66\cdot10^{-3}$		$64\cdot10^{-5}$	$2{,}49\cdot10^{-3}$
4	90	0,174	$11\cdot10^{-3}$		$19{,}2\cdot10^{-4}$	$1{,}83\cdot10^{-2}$	0,087	0,022		$19{,}2\cdot10^{-4}$	0,0145
2	180	0,700	$5{,}5\cdot10^{-3}$		$3{,}86\cdot10^{-3}$	$5{,}25\cdot10^{-2}$	0,35	0,011		$38{,}6\cdot10^{-4}$	0,367
1	360	2,78	$2{,}78\cdot10^{-3}$		$7{,}72\cdot10^{-3}$	$4{,}64\cdot10^{-3}$	1,39	0,0055		$7{,}69\cdot10^{-3}$	0,103
0,5	720	11,12	$1{,}39\cdot10^{-3}$		$15{,}4\cdot10^{-3}$	0,412	5,56	0,0027		$15{,}38\cdot10^{-3}$	0,289
0,1	3600	279,0	$2{,}78\cdot10^{-4}$		$77{,}2\cdot10^{-3}$	4,67	139,5	0,00055		$76{,}9\cdot10^{-3}$	3,27
		$\beta = 5°$					$\beta = 7°$				
360	1	$2{,}15\cdot10^{-6}$	10,0	A	$21{,}5\cdot10^{-6}$	$6{,}82\cdot10^{-6}$	$1{,}53\cdot10^{-6}$	14,0	B	$21{,}5\cdot10^{-6}$	$5{,}72\cdot10^{-6}$
180	2	$8{,}6\cdot10^{-6}$	5,0	C	$43\cdot10^{-6}$	$19{,}2\cdot10^{-6}$	$6{,}15\cdot10^{-6}$	7,0	A	$43\cdot10^{-6}$	$16{,}4\cdot10^{-6}$
120	3	$19{,}4\cdot10^{-6}$	3,3	E	$64\cdot10^{-6}$	$35{,}2\cdot10^{-6}$	$13{,}8\cdot10^{-6}$	4,66	**5** EC	$64\cdot10^{-6}$	$29{,}8\cdot10^{-6}$
72	5	$53{,}6\cdot10^{-6}$	2,0		$107\cdot10^{-6}$	$75{,}8\cdot10^{-6}$	$38{,}2\cdot10^{-6}$	2,8	**2**	$10{,}7\cdot10^{-6}$	$63{,}9\cdot10^{-6}$
60	10	$2{,}15\cdot10^{-4}$	1		$2{,}15\cdot10^{-4}$	$2{,}15\cdot10^{-4}$	$1{,}53\cdot10^{-4}$	1,4		$2{,}15\cdot10^{-4}$	$1{,}81\cdot10^{-4}$
12	30	$1{,}94\cdot10^{-3}$	0,33		$64\cdot10^{-5}$	$1{,}11\cdot10^{-3}$	$1{,}38\cdot10^{-3}$	0,466		$64{,}0\cdot10^{-5}$	$9{,}42\cdot10^{-4}$
4	90	$17{,}4\cdot10^{-3}$	0,11	**4**	$19{,}2\cdot10^{-4}$	$57{,}7\cdot10^{-4}$	$12{,}42\cdot10^{-3}$	0,156	**4**	$19{,}2\cdot10^{-4}$	$4{,}92\cdot10^{-3}$
2	180	$70\cdot10^{-3}$	0,055	D	$3{,}85\cdot10^{-3}$	$164\cdot10^{-4}$	$50\cdot10^{-3}$	0,078	D	$3{,}8\cdot10^{-3}$	$13{,}9\cdot10^{-3}$
1	360	$278\cdot10^{-3}$	0,0278		$7{,}72\cdot10^{-3}$	$46{,}43\cdot10^{-3}$	$198\cdot10^{-3}$	0,038		$7{,}72\cdot10^{-3}$	$38{,}6\cdot10^{-3}$
0 5	720	1,112	0,0139		$15{,}4\cdot10^{-3}$	$13{,}1\cdot10^{-3}$	0,794	0,019		$15{,}4\cdot10^{-3}$	0,109
0,1	3600	27,90	0,0027		$77{,}2\cdot10^{-3}$	1,44	19,99	0,0039		$77{,}2\cdot10^{-3}$	1,24

Z sec.	ω_0 grad.	$\beta = 10°$ $aq = v$ cm./sec.	T sec.		vT cm.	$v\sqrt{T}$	$\beta = 30°$ $aq = v$ cm./sec.	T sec.		vT cm.	$v\sqrt{T}$
360	1	$1{,}08\cdot10^{-6}$	20		$21{,}5\cdot10^{-6}$	$4{,}83\cdot10^{-6}$	$3{,}6\cdot10^{-7}$	60		$21{,}5\cdot10^{-6}$	$2{,}78\cdot10^{-6}$
180	2	$4{,}3\cdot10^{-6}$	10	AF	$43\cdot10^{-6}$	$13{,}6\cdot10^{-6}$	$1{,}43\cdot10^{-6}$	30		$43\cdot10^{-6}$	$7{,}73\cdot10^{-6}$
120	3	$9{,}7\cdot10^{-6}$	6,6	1 C	$64\cdot10^{-6}$	$25{,}0\cdot10^{-6}$	$3{,}23\cdot10^{-6}$	20		$64\cdot10^{-6}$	$14{,}4\cdot10^{-6}$
72	5	$26{,}8\cdot10^{-6}$	4,0	1	$107{,}2\cdot10^{-6}$	$53{,}6\cdot10^{-6}$	$8{,}92\cdot10^{-6}$	12	C	$107\cdot10^{-6}$	$30{,}8\cdot10^{-6}$
60	10	$1{,}08\cdot10^{-4}$	2,0		$2{,}15\cdot10^{-4}$	$1{,}54\cdot10^{-4}$	$3{,}6\cdot10^{-5}$	6	1	$2{,}15\cdot10^{-4}$	$8{,}82\cdot10^{-5}$
12	30	$9{,}7\cdot10^{-4}$	0,66		$64\cdot10^{-5}$	$7{,}88\cdot10^{-4}$	$32{,}2\cdot10^{-5}$	2		$6{,}44\cdot10^{-4}$	$45{,}6\cdot10^{-5}$
4	90	$8{,}7\cdot10^{-3}$	0,22		$19{,}2\cdot10^{-4}$	$4{,}08\cdot10^{-3}$	$2{,}9\cdot10^{-3}$	0,66		$19\cdot10^{-4}$	$2{,}35\cdot10^{-3}$
2	180	$35\cdot10^{-3}$	0,11	D	$3{,}85\cdot10^{-3}$	$11{,}6\cdot10^{-3}$	$11{,}6\cdot10^{-3}$	0,33	D	$3{,}9\cdot10^{-3}$	$6{,}49\cdot10^{-3}$
1	360	$139\cdot10^{-3}$	0,55		$7{,}69\cdot10^{-3}$	$32{,}6\cdot10^{-3}$	$46{,}4\cdot10^{-3}$	0,166	7	$7{,}7\cdot10^{-3}$	$18{,}9\cdot10^{-3}$
0,5	720	0,556	0,027		$15{,}38\cdot10^{-3}$	0,0915	$186\cdot10^{-3}$	0,0835		$15{,}4\cdot10^{-3}$	$53{,}8\cdot10^{-3}$
0,1	3600	13,95	0,0055		$77{,}2\cdot10^{-3}$	1,06	4,64	0,0166		$77\cdot10^{-3}$	0,586
		$\beta = 60°$					$\beta = 90°$				
360	1	$1{,}8\cdot10^{-7}$	120		$21{,}5\cdot10^{-6}$	$1{,}97\cdot10^{-6}$	$1{,}18\cdot10^{-7}$	180		$21{,}5\cdot10^{-6}$	$15{,}8\cdot10^{-6}$
180	2	$7{,}35\cdot10^{-7}$	60		$43\cdot10^{-6}$	$5{,}7\cdot10^{-6}$	$0{,}48\cdot10^{-6}$	90		$43\cdot10^{-6}$	$4{,}35\cdot10^{-6}$
120	3	$1{,}62\cdot10^{-6}$	40		$64\cdot10^{-6}$	$10{,}15\cdot10^{-6}$	$1{,}1\cdot10^{-6}$	60		$64\cdot10^{-6}$	$8{,}56\cdot10^{-6}$
72	5	$4{,}48\cdot10^{-6}$	24	F	$107\cdot10^{-6}$	$21{,}9\cdot10^{-6}$	$2{,}99\cdot10^{-6}$	36		$107\cdot10^{-6}$	$17{,}9\cdot10^{-6}$
60	10	$1{,}8\cdot10^{-5}$	12		$2{,}15\cdot10^{-4}$	$6{,}25\cdot10^{-5}$	$1{,}18\cdot10^{-5}$	18	F	$2{,}15\cdot10^{-4}$	$5{,}01\cdot10^{-5}$
12	30	$16{,}1\cdot10^{-5}$	4	2	$6{,}42\cdot10^{-4}$	$32{,}2\cdot10^{-5}$	$10{,}7\cdot10^{-5}$	6	2	$6{,}43\cdot10^{-4}$	$15{,}4\cdot10^{-5}$
4	90	$1{,}45\cdot10^{-3}$	1,33		$19\cdot10^{-4}$	$1{,}67\cdot10^{-3}$	$9{,}65\cdot10^{-4}$	2,0		$19\cdot10^{-4}$	$1{,}36\cdot10^{-3}$
2	180	$5{,}8\cdot10^{-3}$	0,66		$3{,}9\cdot10^{-3}$	$4{,}7\cdot10^{-3}$	$3{,}9\cdot10^{-3}$	1,0		$3{,}9\cdot10^{-3}$	$3{,}9\cdot10^{-3}$
1	360	$23{,}2\cdot10^{-3}$	0,333		$7{,}7\cdot10^{-3}$	$13{,}3\cdot10^{-3}$	$15{,}4\cdot10^{-3}$	0,5		$7{,}7\cdot10^{-3}$	$10{,}8\cdot10^{-3}$
0,5	720	$90{,}3\cdot10^{-3}$	0,17		$15{,}4\cdot10^{-3}$	$37{,}3\cdot10^{-3}$	$61{,}8\cdot10^{-3}$	0,25		$15{,}4\cdot10^{-3}$	$30{,}9\cdot10^{-3}$
0,1	3600	2,32	0,0333		$77\cdot10^{-3}$	0,424	1,55	0,05		$77\cdot10^{-3}$	0,346

0,1	3600	2,32	0,0333		$77 \cdot 10^{-3}$	0,424	1,55	0,05		$77 \cdot 10^{-3}$	0,346

		$\beta = 180°$					$\beta = 360°$				
Z sec.	ω_o grad.	$aq = v$ cm./sec.	T sec.		vT cm.	$v \cdot \sqrt{T}$	$aq = v$ cm./sec.	T sec.		vT cm.	$v \cdot \sqrt{T}$
360	1	$5{,}99 \cdot 10^{-8}$	360		$21{,}5 \cdot 10^{-6}$	$1{,}13 \cdot 10^{-6}$	$0{,}299 \cdot 10^{-7}$	720		$21{,}5 \cdot 10^{-6}$	$8{,}02 \cdot 10^{-7}$
180	2	$2{,}4 \cdot 10^{-7}$	180		$43 \cdot 10^{-6}$	$3{,}2 \cdot 10^{-6}$	$1{,}2 \cdot 10^{-7}$	360		$43 \cdot 10^{-6}$	$2{,}28 \cdot 10^{-6}$
120	3	$5{,}35 \cdot 10^{-7}$	120		$64 \cdot 10^{-6}$	$5{,}86 \cdot 10^{-6}$	$2{,}68 \cdot 10^{-7}$	240		$64 \cdot 10^{-6}$	$4{,}15 \cdot 10^{-6}$
72	5	$14{,}9 \cdot 10^{-7}$	72		$107 \cdot 10^{-6}$	$12{,}65 \cdot 10^{-6}$	$7{,}45 \cdot 10^{-7}$	144		$107 \cdot 10^{-6}$	$8{,}95 \cdot 10^{-6}$
60	10	$5{,}99 \cdot 10^{-6}$	36	F	$2{,}15 \cdot 10^{-4}$	$3{,}59 \cdot 10^{-5}$	$0{,}299 \cdot 10^{-5}$	72		$2{,}15 \cdot 10^{-4}$	$2{,}53 \cdot 10^{-5}$
12	30	$5{,}35 \cdot 10^{-5}$	12		$6{,}43 \cdot 10^{-4}$	$18{,}5 \cdot 10^{-5}$	$2{,}66 \cdot 10^{-5}$	24	F	$6{,}4 \cdot 10^{-4}$	$13 \cdot 10^{-5}$
4	90	$48{,}2 \cdot 10^{-5}$	4,0		$19 \cdot 10^{-4}$	$56{,}4 \cdot 10^{-5}$	$24{,}1 \cdot 10^{-5}$	8		$19 \cdot 10^{-4}$	$68{,}2 \cdot 10^{-5}$
2	180	$19{,}5 \cdot 10^{-4}$	2,0		$3{,}9 \cdot 10^{-3}$	$2{,}76 \cdot 10^{-3}$	$9{,}7 \cdot 10^{-4}$	4		$3{,}9 \cdot 10^{-3}$	$1{,}9 \cdot 10^{-3}$
1	360	$7{,}7 \cdot 10^{-3}$	1,0		$7{,}7 \cdot 10^{-3}$	$7{,}7 \cdot 10^{-3}$	$3{,}85 \cdot 10^{-3}$	2		$7{,}7 \cdot 10^{-3}$	$5{,}3 \cdot 10^{-3}$
0,5	720	$30{,}9 \cdot 10^{-3}$	0,5		$15{,}4 \cdot 10^{-3}$	$21{,}8 \cdot 10^{-3}$	$15{,}4 \cdot 10^{-3}$	1		$15{,}4 \cdot 10^{-3}$	$15{,}4 \cdot 10^{-3}$
0,1	3600	0,77	0,10		$77 \cdot 10^{-3}$	0,243	0,386	0,2		$77 \cdot 10^{-3}$	0,175

Z = Zeit für 1 Umlauf—Time for 1 revolution in seconds. ω_o = Winkelgeschwindigkeit in °/sec.—Angular velocity °/sec.
aq = v = Strömungsgeschwindigkeit der Endolymphe—Velocity of endolymph. Blank = Unexplored.

A = Threshold of minimum eye deviation after Dohlman.
B = Threshold of minimum eye deviation after Dohlman, but doubtful.
C = Threshold of perception of rotation (Mulder, Dodge, van Rossem).
D = Ordinary clinical rotation test.
E = "Pulsion reflex" after Fischer and Veits; syringing both ears.
F = Light caloric test (Schmaltz).

1 = Rolling of ship (good weather).
2 = Rolling of ship (bad weather).
3 = Automobile on bad road. Oscillation around cross axis.
4 = Automobile turning around street corner.
5 = Railway train on curve.
6 = Railway train on sloping rail in curve.
7 = Sudden wheeling round of head (Buys).

Dr. Schmaltz's exposition, however, had been beautifully clear, even to a non-mathematical person like himself.

The PRESIDENT said that Members were indebted to Dr. Schmaltz, not only for the most careful preparation and the interesting contents of his paper, which must have entailed very considerable trouble and expense, but also for his consideration in delivering his lecture in such extraordinarily correct English.

Dr. Schmaltz's physical investigations seemed to confirm the efficiency of the clinical value of Kobrak's minimal caloric test. Dr. Schmaltz had suggested that the element of fatigue should perhaps also be taken into consideration, a point with which probably all Members would agree. On the other hand, Professor Quix, of Utrecht, had shown him (the President) an apparatus by which it was possible for continuous irrigation of the ear to be maintained for a long period—he understood for two or three hours—and that during this time a continuous effect could be demonstrated as represented by a continuous nystagmus. ("Ein Apparat zur Untersuchung des Vestibularapparates," F. H. Quix, Utrecht; as described at the Twenty-first Meeting of the German Otologists in Hanover, May 1912).

He (the President) had been rather puzzled that Dohlman had found (as recorded in one of Dr. Schmaltz's illustrations) that the threshold for sensation of rotation was higher than the threshold for the objective nystagmus. He himself had always supposed that they appeared in the reverse order, and indeed the psychic element, he thought, must always be a factor in these phenomena, since it was common to find a complaint of dizziness in patients in whom no spontaneous nystagmus could be seen.

He would like to ask Dr. Schmaltz if he had any opinions to offer in connection with the reports of van Wulfften Palthe, who had investigated the sensations of a blindfold passenger in an aeroplane, with the result that under these circumstances the sense of position in space was apparently totally lost "Function of the deeper sensibility of the vestibular organs in flying," *Acta Oto-Laryngologica*, vol. iv, p. 415).

Dr. SCHMALTZ, in reply to the President, said he agreed that the psychic element entered into the problem too. As far as the stimulation of the labyrinth in an aeroplane was concerned the movements up and down had of course little to do with the stimulation of the canals, as such movements were perceived by the otoliths. If the machine were bending or inclining in taking a curve (sketch) the force acting upon the inner ear in relation to the axial direction of the pilot's body would remain the same, because the resultant force of the gravity and the centrifugal force were always in the sagittal direction of the aeroplane and the pilot sitting in it. A Dutch observer declared that if the aeroplane were flown in a fog, so that the correction by the eyes was lost, the pilot might even be flying upside-down without being aware of the fact.

Referring to another point made by the President, as to the threshold for the sensation of rotation being higher than that for objective nystagmus, that had puzzled the speaker also. But the figures of Dohlman referred to the very smallest deviations of the eye, a matter of an angle of a few minutes, the measurement of which could only be made photographically.

In reply to Dr. Mackenzie he agreed that these occurrences could not be explained purely on a physical basis, and he did not advance that contention: indeed, he had definitely made this statement in an early part of his address. Biological effects in the central nervous system must also be taken into account. On the other hand, he felt very sure that the physical phenomena occurred in the way he had indicated. Exactly how far physical agency was responsible for these phenomena was another question.

As to the small calibre of the tube, in the older literature it was a subject of much discussion whether the fluid could flow in a tube of such a minute calibre. But, from experiments which had been made by means of glass canals of much smaller diameter than the human semicircular canals, he could positively say liquid would flow, and would obey the ordinary mechanical laws applicable to the movement of fluids in tubes (Law of Poiseuille). The flow of endolymph in response to the caloric test had been observed by Maier and Lion (in the clinic of Prof. Voss, Frankfurt), who opened the bony canal of a pigeon and stimulated the labyrinth thermically, and, by means of the microscope, they saw the red cells in the endolymph moving along.[12] With regard to the part the utriculus

[12] Maier and Lion, *Pflüger's Archiv f.d. ges. Physiologie*, 1921, clxxxvii, 47.

played in the scheme experiments and theory showed that, as far as the movement of the endolymph was concerned, it made very little difference that the semicircular canal terminated in a large cavity such as the utriculus. It was necessary to distinguish between the laminal flow, in which all the different parts of the liquid remained parallel with each other, and what is called turbulent flow. Reynolds had shown that there was a limit of speed, and when that was exceeded there was no more laminal movement, there was turbinal movement. But, as the speed of the endolymph was so small there were certainly laminal movements, and in this case we could feel sure that the influence of the utriculus upon the flow of the endolymph was very small and the ideal model of a closed circular canal which the author had supposed when putting down his theory was sufficiently correct. L. d. Nó had also investigated the influence of the utriculus upon the movement of the endolymph and had come to the same result, namely, that this influence was very small.